DETAILED ANALYSIS OF ANTEMORTEM BURNS IN RELEVANT TO CARBOXY HAEMOGLOBIN

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CERTIFICATE

This is to certify that the dissertation entitled "***DETAILED ANALYSIS OF ANTEMORTEM BURNS IN RELEVANT TO CARBOXY HAEMOGLOBIN***" is the bonafide work of **Dr. K.RAJAVELU** in partial fulfilment of the university regulations of the Tamilnadu Dr. M.G.R. Medical University, Chennai, for M.D., (Foresic Medicine) Branch–14 examination to be held in April 2013.

Prof. Dr.G.Natarajan. M.D.,
Head of the Department,
Department of Forensic Medicine,
Madurai Medical College,
Madurai.

DECLARATION

I, **Dr. K .RAJAVELU**, hereby declare that, I carried out this work on **"Detailed Analysis of Antemortem Burns in relevant to Carboxy Haemoglobin"** at the Department of Forensic Medicine, Government Rajaji Hospital, Madurai, under the guidance of **Prof. Dr. G. Natarajan, M.D.,** Head of the Department of Forensic Medicine, during the period of October 2011 to December 2012. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other University or Board either in India or Abroad.

This is submitted to the Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfilment of the rules and regulations for the M.D. Degree Examination in Forensic Medicine (Branch –14) to be held in April 2013 .

(Dr.K. RAJAVELU)

Place : Madurai

Date :

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CONTENTS

INTRODUCTION

Differentiation of antemortem burns from postmortem burns is a difficult task and also medico legally important one. It is mostly a matter for the police investigation.

After homicide, attempts are made to burn a body ,with an aim to concealing the crime. If a burned body, discovered in an unfamiliar environment and abandoned place , it give suspicion about the cause of death. Presence of accelerant used and absence of vital signs are indicators of postmortem burning following murder. In such cases the body should be explored for marks of violence like stab wounds, bullets, strangulation etc.

At present there is no definite sign, to differentiate the antemortem and postmortem burns. Although a few criteria can be used as **indicators** of antemortem burns such as presence of **soot particles in the respiratory tract, vital reaction around the burnt area and the carboxy haemoglobin in the bood .**

Regarding soot particles it may be absent in imminent death.On histological examination, if there is demonstration of soot in the terminal bronchiole, that is an absolute proof of respiratory function.

Because of heat irritation of tracheal and bronchial walls, the carbon is usually mixed with mucus.

Often there is swallowed soot and mucus in the stomach. This is again an evidence of life during the smoky phase of the fire.

Like carbon monoxide, victim of fire usually breathe in carbon particle present in the sooty smoke. It is more common in conflagration than a vehicle blaze.

Large volume of dense smoke – produced by the burning of

- Fabric of furniture
- Carpets.
- Timber of floor, roofs and furniture.

As a marker of ante mortem burns – it is almost as useful as carbon monoxide, but in some cases soot enters the open mouth after death, but it cannot enter the trachea (or) bronchi. Soot, only stain the mucosa of tongue and pharynx, may even passively reach the glottis but no

significant amount of soot enter the vocal cords and enter the trachea after death.

So carbon in the lower respiratory tract , is an indicator of breathing, during fire.

Regarding vital reaction, it is absent if the whole body is burnt and it can also produced if the body is burned within one hour after homicide. Burns produced shortly before or after death cannot be distinguished either by naked eye or by microscopic examination. So burns produced during the perimortem period because of reddening, creates confusion regarding cause of death.

Regarding carboxy haemoglobin it is proved by the previous studies, that it is definitely absent in all the postmortem burns.

In previous studies, the carboxy haemoglobin is **positive** in some of the antemortem burns and was **negative** in all of the postmortem burns.

Here an attempt has been made to study the prevalence of carboxy haemoglobin in death due to burns. This study tries to find out, any relationship between carboxy hemoglobin level in blood and sex, age, fire

accelerants used and the environment in which the incident occur. Carbon monoxide concentration in blood, depends on the following factors.

- Concentration of carbon monoxide in the inhaled air.
- Duration of exposure
- Status of respiration.(rate and depth).
- The hemoglobin content of the blood.
- Activity of the victim.

Possible reasons for absence of carboxy hemoglobin in the blood.

- Imminent death.
- Convection air currents.
- Low production of carbon monoxide.
- Flash fire.
- Super heated air that cause suffocation on inhalation. (in warfare)
- Explosive burns.

The carbon monoxide is the most common asphyxiant that cause inhalation injuries in case of burn. Carbon monoxide is released during

the incomplete burning of all organic materials, most commonly presence of wood in the fire.

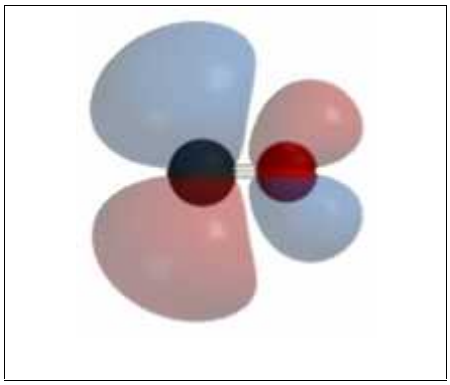
When the head has been exposed to intense heat, sufficient to cause charring of the skull, produce heat hematoma. It has the appearance of extradural hemorrhage. In this type of cases, carboxy hemoglobin helps to differentiate between this two. The heat hematoma will contain carboxy hemoglobin, but extradural hemorrhage caused by injury before the fire began will not show carboxy hemoglobin.

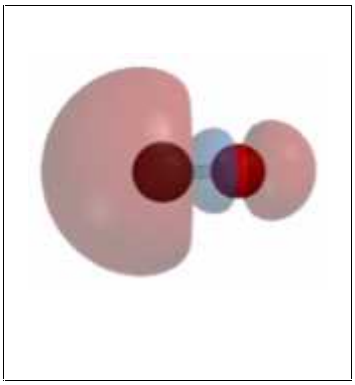
The features that helps to differentiate antemortem burns from postmortem burns.

SL. NO	TRAIT	ANTEMORTEM BURNS	POSTMORTEM BURNS.
1.	Line of redness	Present all around	Absent.
2.	Blister.	More fluid than gas. Fluid contains more protein and less chloride.	More gas than fluid. More chloride, less protein.
3.	Soot particles in the air passage	Present in trachea. Even present below the bifurcation.	Not present.
4.	Base of the blister	Congested.	Absent.
5.	Enzymatic reaction.	Present in and around the burns.The	No specific change.

		reaction is less in the centre than the peripheral zone.	
6.	Reperative process.	Present.	Absent.
7.	Blood	Cherry red due to Co.	Not so.
8.	Curling's ulcer.	May be seen.	Absent

Figure 1 :





AIMS AND OBJECTIVES

1. To identify the presence of carboxy haemoglobin in death due to burn.

2. To identify whether any association exists between age, sex, place of occurrence, fire accelerants used **and presence** of carboxy heamoglobin in blood.

REVIEW OF LITERATURE

Asphyxia that is produced by carbon monoxide is due to it's high affinity for haemoglobin than oxygen. There by affect the transport of oxygen to the tissue, which in turn results in tissue hypoxia. Because of it's high affinity, it produce significant cell damage even in low concentration in air. Person affected by carbon monoxide poisoning can be recovered if treated in time. But in case of acute poisoning, even if the person recovered , results in permanent damage to the organs that receive lot of oxygen eg., (heart and brain).In the managemen of carbon monoxide poisoning, affected person must be moved to the fresh air in open area .Administration of 100%Oxygen helps in early recovery in most of the cases. Cardio pulmonary resuscitation required in some cases.

Tissue hypoxia in turn affects the oxidative phosphorylation and produce cellular injury.

PRESONS IN RISK OF CARBON MONOXIDE POISONING :-

- Marine terminal worker.
- Taxi driver.
- Police officer.
- Fire fighter

In case of fire, oxygen concentration in the environment reduced to as low as 2% from the normal level of 21%.It is because the burning materials utilize the oxygen that is present in the environment.

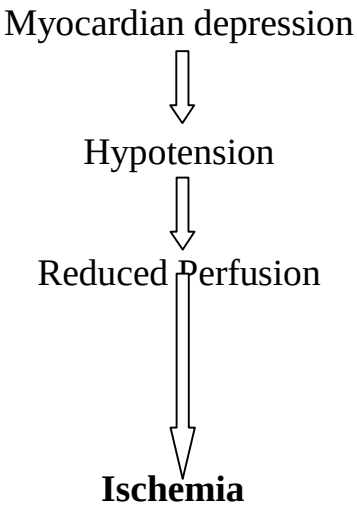
Transport of oxygen affected

Tissue hypoxia.

Cellular injury

So both the low oxygen concentration and binding of carbon monoxide with the haemoglobin produce more injury to the cells by causing anaemic hypoxia. But the binding of carbon monoxide with haemoglobin is a reversible phenomenon.

In addition to this, carbon monoxide also bind with the myoglobin of cardiac muscle with the greater affinity. Myoglobin is saturated by carbon monoxide in three times higher than skeletal muscle. This affects the cardiac function by the following way.



that potentiate the hypoxia induced by decreased oxygen delivery.

Carbon monoxide that bind with the cytochrome c and p450 but with a lower affinity than oxygen. Co also acts on cytochrome oxidase and reduce the level. There by cause inhibition of cellular respiration. So it is difficult to assess the toxicity of the carboxy haemoglobin level in the blood.

Cellular action of carbon monoxide is depends on it's concentration and the concentration of molecular oxygen, presence of transition metals such as Fe(II).These factors influence local oxidation – reduction reaction, there by determine the physiological effect of carbon monoxide. It also influence the physiological effects of nitric oxide.

Carbon monoxide poison affect the heart and brain by anemic hypoxia. It is important to differentiate the effects produced in arterial hypoxemia and myocardial ischemia on the heart. Both are differ in the following aspects- arterial hypoxemia is generalized, but myocardial ischemia is localised. Cellular metabolism of ischemic myocardium differ from that of hypoxic myocardium.

Changes in myocytes-

- Decline in ATP concentraion
- Increased glucose uptake
- Increased anareobic glycolysis..
- Pyruvate formation.
- Lactate and alanine formation.

Hypoxic changes in myocardium

Hypoxia

⇩

Direct and Reflex effect

⇩

Vasodilatation of cerebral and coronary blood vessels.

⇩

Stimulation of carotid body chemoreceptors.

⇩

Reflex vasoconstriction of splanchnic, muscle,
Pulmonary and cutaneous beds, with or without net
effect in the coronary and cerebral beds.

Carbon monoxide produce anaemic hypoxia, there by affects particularly the neurons of brain, because the neurons are susceptible particularly to hypoxia since they have obligative aerobic glycolytic metabolism.

Adult brain receives 15% of cardiac output or 45ml/100mg of tissue/minute. The respiratory quotient of the brain is almost unity and the glucose is the principle source of energy by oxygenation.

Breathing of hypoxic gas cause, increased coronary blood flow. Reduction of the 10% oxygen concentration in the inspired air, results in doubling of coronary blood flow because of decreased coronary arterial oxygen content.

It have not been reported that, change in coronary blood flow to, hypoxia of other clinical situation. In lungs hypoxia, causes increased pulmonary vasculature resistance when the Po2 less or equal to 8KPa.There is wide individual variation.

In case of hypoxia, the oxygen or glucose level is reduced below the critical level ,results in loss of consciousness after a few seconds and if prolonged it may results in irreversible brain damage.

SUPPLY OF OXYGEN TO THE BRAIN DEPENDS ON

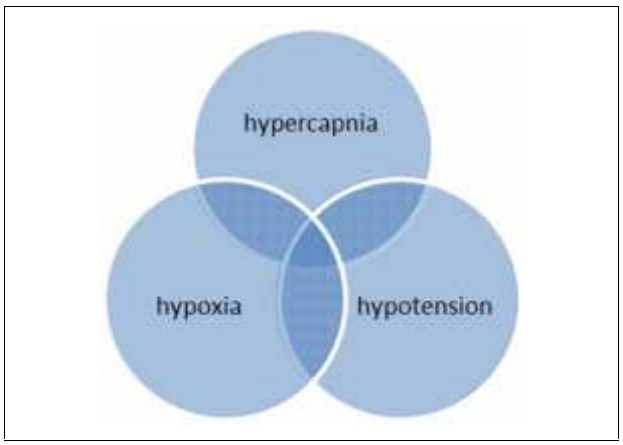
Cerebral Blood Flow Cerebral Perfusion Pressure.

(Difference between the mean systemic arterial pressure and cerebral venous blood pressure.)

Blood flow to the brain has the remarkable capacity for remaining constant. But it is affected by

- Hypercapnia.
- Hypoxia.
- Extreme hypotension.

Figure 2 :



But preservation of cerebral blood flow, in response to change in arterial blood pressure is brought about by “autoregulation”.

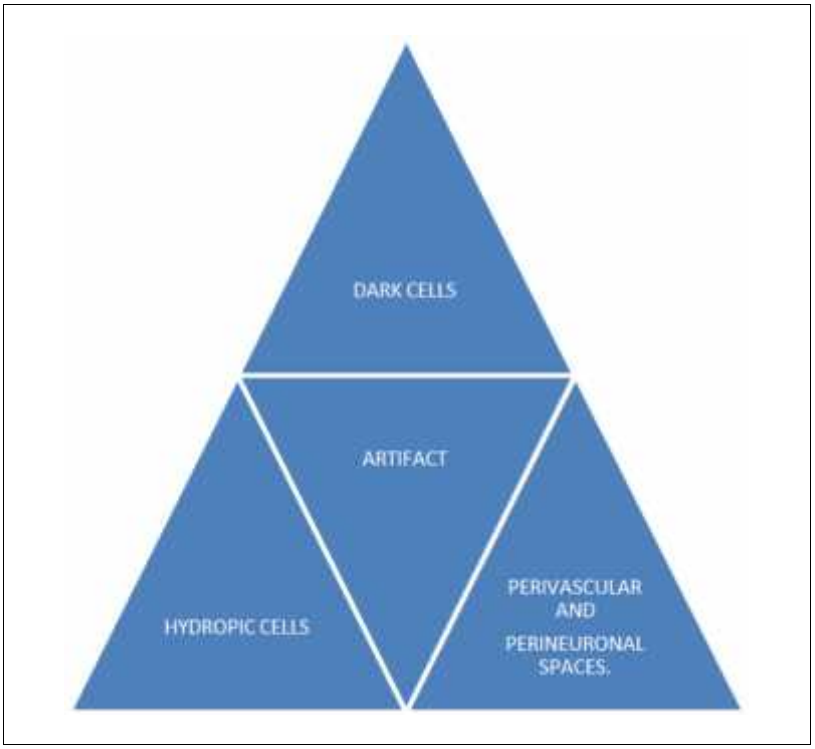
The mechanism of this auto regulation is still uncertain because it is appear to be lost or severely impaired in a wide range of acute conditions producing brain damage.

There are many situations in which cerebral auto regulation may be impaired before an episode of hypoxia. This is the fact that supports why it is not possible to detect hypoxic changes in all the case of carbon monoxide poisoning occurs in smoke inhalation.

It is difficult to find out the ischemic cell changes in the human brain because of the consequent appearance of histological artifact.

Following are the artifact which is common in such a examination.

Figure 3 :



- DARK CELLS
- HYDROPIC CELLS
- PERINEURONAL AND PERIVASCULAR SPACES.

These artifacts are partly due to post mortem handling and slow penetration of fixative.

HISTOLOGICAL CHANGES IN HYPOXIA.

HISTOLOGICAL CHANGES IN HYPOXIA .

MICROVACULATION
ISCHEMIC CELL CHANGE.
INCRUSTATION.
AND
HOMOGENOUS CELL CHANGE.

1. Microvacuolation

   it is proved only in animal brain.

2. Ischemic cell change.

   Cell bodies and nucleus become shrunken.

   Cytoplasm has been stain intensely with eosin and crystal violet. Nucleus has been stain with basic aniline dyes.

3. Incrustation.

   There is further shrinkage of nerve cell cytoplasm. Small and dense granules seen close to the surface of nerve cell.

4. Homogenous cell change.

Most commonly seen in purkinji cells of the cerebellum. The cytoplasm becomes homogenous. Nucleus appears smaller.

THE SUSCEPTIBILITY OF NERVE CELLS TO HYPOXIA IN DESCENDING ORDER.

Nerve Cells

⇩

Oligoden Troglia and Astrocytes

⇩

Microglia and Cellular Elements of Blood Vessels

ANAEMIC BRAIN DAMAGE: This is a condition that occurs in carbon monoxide poisoning. The most common complication arise from nervous system. If the death happen within few hours, the organs appear pink in color.

In case of carbon monoxide poisoning, if the death occurs within 36 to 48 hours , there is congestion and petechiae seen in the white matter and the corpus callosum. There is also necrosis of neurons present over the Ammon's horn and the cerebellar and cerebral cortex.

Most of the changes seen in the white matter .Damage to white matter specifically develop in person, with delayed signs of intoxication with a interval of normal period ,after acute poisoning.

The level of cerebral perfusion pressure, that cause damage to brain is not known in human being, but in the presence of normal auto regulation the critical level of systemic arterial pressure to prevent the brain damage is 50mm.Hg. In person with normal paO2, it would appear that brain damage does not occur, until the CPP falls to less than 25mm.Hg.

CATEGORIES OF BRAIN HYPOXIA

- Stagnant. (a).Ischemic.(b).oligaemic.
- Anoxic and Hypoxic.
- Anaemic hypoxia= occur in carbon monoxide poisoning, in which there is insufficient hemoglobin in the blood, to carry the Oxygen in chemical combination.
- Histotoxic- poisons of neuronal respiratory enzyme.
- Hypoglycemic- deficiency of substrate glucose.
- Febrile convulsions and status epilepticus.

The effect of local exposure of the body, to heat is, to produce a burn, the severity of which depends on the length of exposure and the temperature of the heat source.

The minimum temperature capable of producing burn is ,above 44 degree centigrade, for an exposure of about 5 to 6 hours. 2 second is sufficient at 65 degree centigrade.

A dead body of course be burnt ,as well as live one. Post mortem burns can be distinguished from antemortem injuries, by the absence of reddening of skin at the margins, the absence of significant amount of protein in the fluid of blisters, and the absence of microscopic evidence of inflammatory reaction.

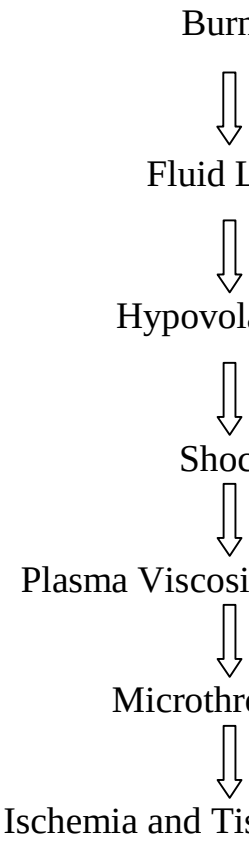
Differentiation of burns ,whether it is accidental or suicidal or homicidal is mostly a matter for the police investigation.

This usually arises, when a body is found in a house or vehicle which has been the seat of fire. The same type of consideration apply as in the case of drowning,i.e . the fact that the body is burnt, and, at the site of a fire, is not sufficient proof that the death was due to burns. The person may have died naturally, before the fire or have been murdered and the fire started to conceal any evidence of the crime.

When a burnt body is brought for postmortem, the following points have to be noted for

- If the body is charred beyond recognition, features of identity.
- Examination of clothing and personal articles. Useful to find out the fire accelerant.
- Description of nature and extent of burns.
- Presence of ante mortem injuries other than burns.
- Examination of respiratory passage for suit particles.
- Spectroscopic/gas chromatography examination of blood for the presence of carboxy hemoglobin.
- Chemical analysis of viscera, blood and urine for the presence of carboxy hemoglobin.
- In delayed deaths, histological examination of kidney, brain, etc for renal damage, septicemia and fat embolism.

PATHOPHYSIOLOGY OF BURNS:-



Large numbers of burn death are accidental. Accidental burn death may occur in the following circumstances.

- Inside the kitchen. Clothes of the women catch the fire accidentally. The features of this burns are-
    1. Burns are usually found on the front of the thigh, abdomen, chest and face.
    2. Severe burning of hands due to the victim trying to extinguish the fire by beating out the flames.
    3. Feet and ankles are usually not burnt.

- In factories..Explosion from the inflammable liquids and flashes from furnaces.
- House conflagration.
- Flaming of highly inflammable fuel.
- Electrical short circuits.
- Manufacture and playing with fireworks.
- Multiple burn death in plane crashes and in automobile accident.
- Leakage of cooking gas.
- Explosion of lamp or stoves.
- Intoxicated person goes to bed with smoking cigarette.

SPONTANEOUS COMBUSTION: At one time the body was thought to be relatively incombustible, due to its high water content. However, experiments shown that human fat is quite inflammable. This accounts for the occasions when a dead body, ignited at one point by a relatively small heat source , such as domestic hearth fire , becomes almost completely consumed within a short time , though objects in a close proximity to the body e.g. furniture in a room , are not damaged.

These circumstances are known as preternatural combustion. In the old days they were ascribed by the credulous to “spontaneous combustion” or to mystical events .

Death in a burning building may be due to

- Burns.
- **Poisoning by inhaling fumes, notably carbon monoxide.**
- Injuries from falling masonry, etc.
- Natural disease, e.g. Heart failure due to coronary atheroma, precipitated by fear or exertion.

SUCIDAL BURNS:-

More common in women than man. They commit suicide by pouring kerosene on their head and clothes before setting fire to themselves. Extensive first and second degree burns, more concentrated on the front of the body are seen all over the body. Only the skin folds like axillae and perineum and soles being spared.

HOMICIDAL BURNS;-

Murdered by burning is a rare one.

If a inflammable fluid, poured on a person lying on the back and then burnt, there will be burning of the

- Sides of the neck.
- Sides of the trunk.
- Between the thighs and other areas as the fluid run downwards. Parts of the body which are in contact with ground do not show burning..

Concealment of a crime by arson:

Examination of the body may reveal bullet wounds, stab wounds, ligature round the neck, etc., and no signs that the diseased was alive during the fire. It is unlikely that such evidence of crime will be destroyed unless damage by burning is very severe.

Presence of carbon particles and elevated Carboxy haemoglobin level are absolute proof that the victim was alive when the fire occurred. Some of the factors that influence the effects of burns include

- Intensity of the heat.
- Duration of exposure.
- Depth of the burns.

Depends on the skin and it's morphology burns classified in to three types

- Superficial burns: Affects the epidermis and characterized by redness and blisters.
- Partial thickness burns: Extends into dermis. At mid dermal level, it is pink in color with small white patches and painful. If it is deep appears white and does not blanch on pressure and also less sensitive to pain.
- Full thickness burns: Appears white and painless. Does not blanch on pressure. Scars and contracture on healing.

In addition to the depth of burn, the other factor of importance is the surface area of the body which is affected. Twenty percent involvement, even by superficial burns, is dangerous to life.

In a case of burns, calculation of (BSA) Body Surface Area(burned) helps in estimating the prognosis and to decide the line of treatment. The calculation is done by using Rule of Nine. If the BSA is 20% indicates marked fluid loss. If it is 30 to 50% , that denotes the fatal outcome. But in children Lund and Browder chart used to calculate the BSA.

RULE OF NINE

Anatomical area.	Percentage of total body area
Head and neck (front and back)	9%
Right upper limb.	9%
Left upper limb.	9%
Front of the chest.	9%
Back of the chest.	9%
Front of the abdomen.	9%
Back of abdomen.	9%
Front of Right leg.	9%

Back of right leg.	9%
Front of left leg.	9%
Back of left leg.	9%
Perineum and external genitalia.	1%

FOR CHILDREN

Anatomical area	Percentage of total body area
Head and Nck	18%
Chest and Adomen (front)	18%
Chest and Abdomen (back)	18%
Upper Lmb.	9% + 9%
Lower Lmb.	13.5% + 13.5%
Perineum.	1%

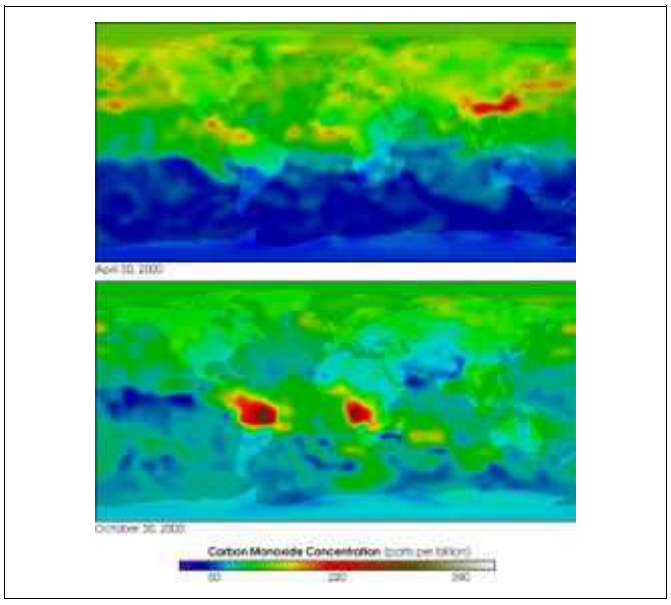
Postmortem finding, in a case of burn death, due to carbon monoxide inhalation.

- Cherry red discoloration of skin, mucous membrane, postmortem lividity, blood and viscera
- Fine froth at nostrils/mouth
- Lungs :-Edema and congestion

- Heart:-Lesion varies from petechial hemorrhage to Myocardial necrosis.
- Globus pallidum and putamen of basal ganglia shows necrosis and cavitations.
- Blisters of skin over the pressure points such as calves, wrists, buttocks and knees or dependant areas.
- Rhabdomyolysis from the toxic effects of carbon monoxide and prolonged immobility lead to renal failure.

CARBON MONOXIDE IN THE ATMOSPHERE .

Figure 5:



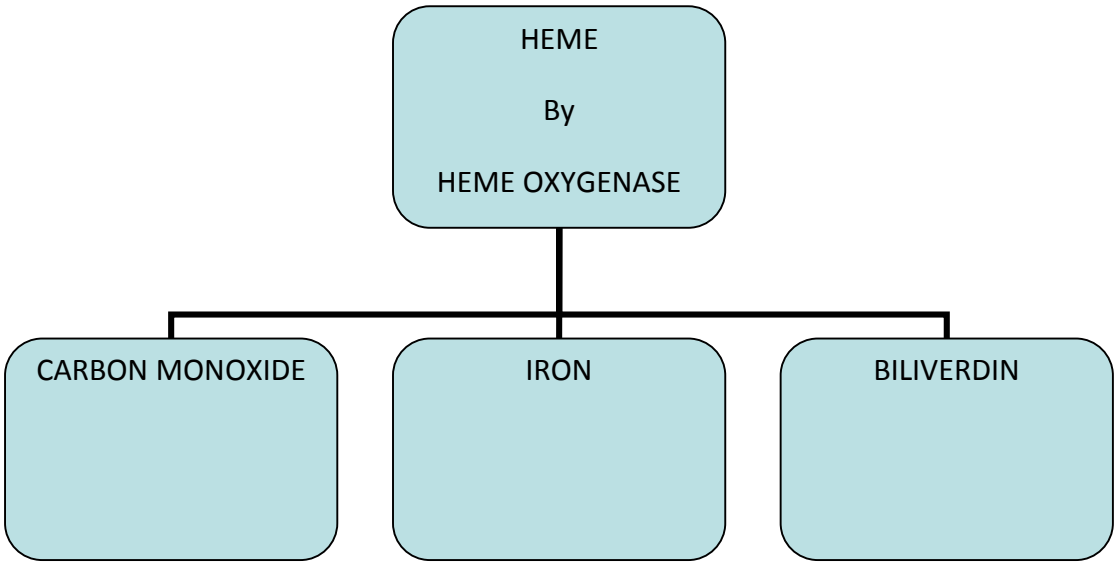
Carbon monoxide poisoning occurs after enough inhalation of carbon monoxide (CO). Carbon monoxide is a toxic and colorless, odorless, tasteless gas. Because it is non-irritating, initially it is very difficult to detect by the people, those who are involved in the rescue operation in case of fire accident. . Exposures at 100 PPM is dangerous to human health.

So it is advisable for rescue people to crawl along the ground in a conflagration.

Properties of Carbon monoxide.

It is produced naturally in the human body in harmless range and helps in physiological function including vasodilatation, vascular remodeling,angiogenesis and modulation of inflammatory process.

Among the carbon monoxide produced 85% produced by heme oxygenase



The major site of heme catabolism is in liver . so the major site of carbon monoxide synthesis is in liver. The normal level of blood carboxy hemoglobin in nonsmokers is approximately 1%.

If a person takes 20 cigarettes per day the mean saturation of carboxy hemoglobin is 5.5%. The majority of carbon monoxide is removed by expiration.

There are two isomers of heme oxygenase. They are HO-1 and HO-2.

Among these two, HO-1 is the only inducible form.It is induced by hypoxia, heavy metals, sodium arsenite, heme and heme derivatives, oxidative process as well as by cytokines.

In patients with following condition there is increased expression of HO-1 and elevated carboxy hemoglobin.

- **Chronic obstructive pulmonary disease.**
- **Systemic inflammatory response syndrome.**
- **Acute respiratory distress syndrome.**

HO-1 induction is beneficial because the the products have anti inflammatory and anti oxidant properties.

So in case of cardiothorcic surgery the patient more likely to die on lower minimum or higher maximum CoHb level.

Activity of HO-1 if excessive is deleterious,possibly due to the liberation molecular iron.

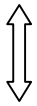
In the previous studies , even low levels of CoHb decrease the exercise tolerability and also produce cardiac arrhythmias in

non smokers with coronary artery disease .So with the reports from the previous studies, we came to know there is individual variation in the response to the CoHb level.

Oxygen combined with carbon, and form two type of gases. If there is plenty of oxygen and complete combustion there is formation of carbon dioxide . It is heavier than air.

In case of incomplete combustion, because of insufficient oxygen, cause the formation of carbon monoxide. It is lighter than air.

SOURCE OF CARBON



COAL, COKE AND CHAR COAL

Carbon monoxide is formed while burning of natural gas, petrol and diesel. The amount of carbon monoxide produced depends upon the efficiency of combustion.

PREPERATION OF CARBON MONOXIDE IN THE **LABORATORY**

STEP-1

To produce carbon monoxide in the laboratory, source carbon dioxide is needed. Source of carbon dioxide .

1. Carbon dioxide Cylinder
2. Dry Ice.

If it is available, first carbon dioxide is produced by combining acid and hydrogen carbonate or acid and a carbonate.

$$2Hcl + CaCO_3 \longrightarrow CaCl_2 + H_2O + CO_2.$$

By this way CO2 produced. Then it is passed over the heated charcoal. This facilitate the formation of CO.

$$2NaOH + CO_2 \longrightarrow Na_2CO_3 + H_2O$$

STEP2 :

By dehydrating the methanoic acid using concentrated sulphuric acid we get the carbon monoxide.

$$HCOOH \longrightarrow 2CO + 4H_2O.$$

By dehydration of sodium methanoate also we get CO. In this method h2so4 concentrated and dropped directly in to the solid. the carbon monoxide formed is collected in to the water. In a situation where there is incomplete combustion of natural gas the following type of reaction occurs.

$$2CH4 + 3O2 \longrightarrow CO + H_2O$$

Before the discovery of natural gas, for burning, coal gas was used. The coal gas contain methane, hydrogen and carbon monoxide. Coal gas was produced, when it was heated in the absence of air.

As a strong reducing agent carbon monoxide act within the blast furnace. It reduces metal oxides.

$$FeO_3 + 3CO \Longrightarrow 2Fe + 3CO_2$$

REACTION OF CARBON MONOXIDE **:**

$CuO + CO \Longrightarrow Cu + CO_2.$

$CuO + C \Longrightarrow Cu + CO.$

$C + O_2 \Longrightarrow 2CO.$

$C + O_2 \Longrightarrow CO_2.$

$CO_2 + C \Longrightarrow 2CO.$

Co have affinity for transition metals. Co also have acidic or basic properties.

Co reacts with nickel so quickly etched to the surface and highly toxic.

At 192 degree centigrade carbon monoxide can be condensed to liquid. Carbon monoxide also freezes at 199 degree centigrade.

Physical properties of carbon monoxide closely resemble that of nitrogen. At high temperature carbon monoxide with water vapors forming carbon dioxide and hydrogen. By adding the resultant hydrogen with nitrogen used in the synthesis of ammonia.

Carbon monoxide reacts with alkali formats form formic acid and then oxalic acid. Carbon monoxide with metals form carbonyls which are volatile. Carbon monoxide with hydrogen, used as a initial material in the production of methanol. Also used in the making of alcohol and aldehyde. It is in turn used, in the making of mixtures of liquid hydrocarbon in the fuels. **Synthesis gas** contain varying ratios of carbon monoxide and molecular hydrogen.

Abnormalities present in the burns cases, under treatment.

- Oxygen saturation is decreased..
- Metabolic acidosis that reflects the ischemia and hypoxia.
- ECG. Shows ischemic changes.
- EEG. Shows diffuse slow waves and low voltage.Features of hypoxic encephalopathy.
- Chest X-Ray: 1. Ground glass appearance. 2. Peribronchial cuffing. 3.perihilar haze and 4. intra alveolar edema.
- CAT Scan. It is one of the investigation which predicts accurately the neurologic sequlae within 24 hours.(low density globus pallidus).

Magnetic Resonance Imaging;

More effective in detecting tissue edema caused by demyelination in the brain the effects produced by carbon monoxide is cytotoxic edema and demyelination in the white matter and basal ganglia. But these findings are nonspecific because it is also associated with

- cyanide, disulfiram, and hydrogen sulphide poisoning.
- Barbiturate poisoning.
- Hypoglycemia.

Pulse oximeter:- It gives higher reading than the actual oxyhaemoglobin and so there is delay in treatment for hypoxia. It is because, CoHb absorbs the light like oxy haemoglobin at 660 nm. It measures the “functional saturation.”

Co-oximeter: It measures the light absorption at four wave lengths and uses the extinction co efficient at each wave length. It measures the “Fractional saturation”.

Concentration of carbon monoxide in the blood and symptoms produced.

If the blood carboxy hemoglobin level is ten percent – symptomless.

If the level is fifteen percent- mild headache.

If the blood level is, Twenty five percent-severe headache and nausea.

If it is 30 percent- all the symptoms are intensified.

In a situation when the blood level reach 45 percent –the person become unconscious.

Death may occur if the concentration reaches 50 percent or above.

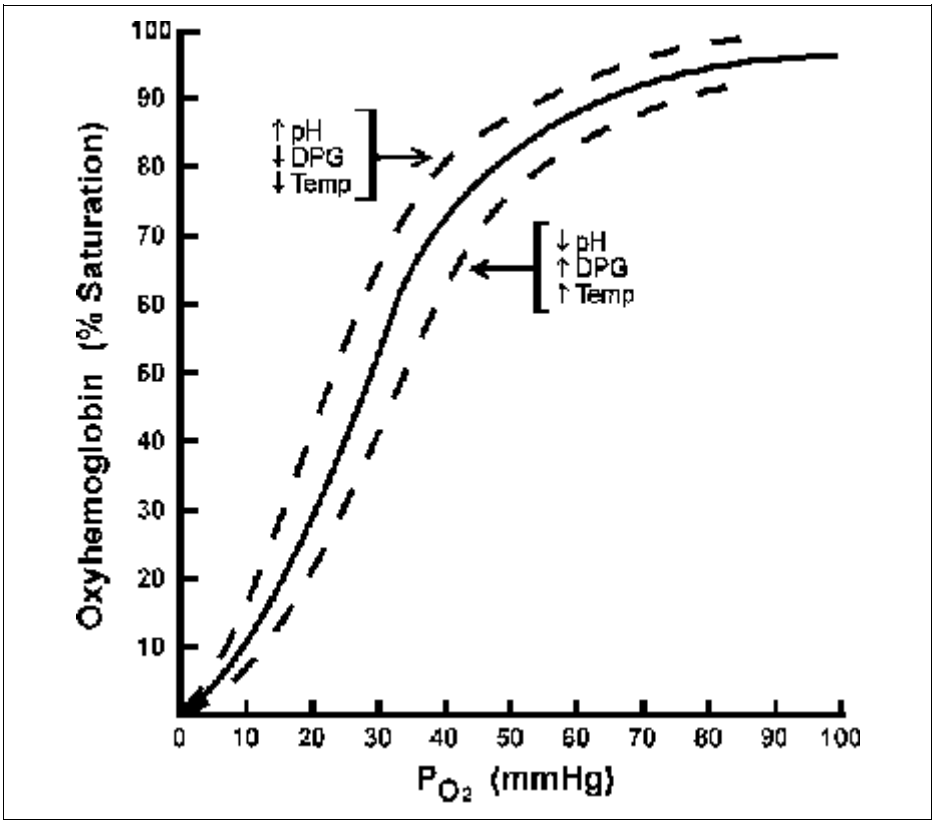
In non smokers usually the Co concentration is less than 3%. In smokers it is between 2 to 10%.

SYMPTOMS ASSOCIATED WITH THE TIME OF EXPOSURE AND COCENTRATION OF CARBON MONOXIDE.

CO (PPM)	TIME	SYMPTOMS
35	8 Hours	Maximum exposure allowed 8 hours.
200	2-3	Mild headache , dizziness and nausea.
400	1-2	Headache become severe.
800	45 Minutes	Convulsion. Unconsicious within 2 hours.
1600	20	death within 1 hour
3200	5-10	death within 1 hour.
5400	1-2	Death with in 25 – 30minutes.
12800	1-3 Minutes	death.

A case report of previous article from Canada , denotes a peculiar case of suicide by complete inhalation of carbon monoxide . In this case the victim is a female , who commit suicide in a closed house by put fire on the surrounding furniture and wait in the corner of that room. So she was planned to die after the inhalation of smoke, from the incompletely burned material, that contain carbon monoxide. On postmortem examination it shows 67% carboxy haemoglobin level.

Figure 6 :



(The American society of Health System)

The graph shows the changes in temperature, ph, organic phosphates like DPG directly affect the dissociation of oxygen.

At the time of fire or in a conflagration the material burned is important .As the toxic substances released was depends upon the material burned.

TOXIC SUBSTANCE	LIBERATED FROM (The burning of)
Nitric acid Phosgene Other complex substance	Furnishings Upholstery Paints Lacquers Varnishes
Cyanide and oxides of nitrogen	Plastic and synthetic material
Nitrogen	Nitrocellulose film
Nitrogen tetroxide	Film
Ammonia Hydrogen cyanide Hydrogen sulphide Oxides of sulphur	Wool or silk

In antemortem burns,the area around the burns show enzymatic reaction, which are more in peripheral zone than central zone.

- ✓ Enzymes involved are SH groups – in all layers.
- ✓ Acid mucopolysaccharides- present in the superticial layer of burnt area .

ENZYME	TIME OF REACTION
ATP and esterases increased in,	1 to 2 hours
Aminopeptidases	2 to 4 hours
Acid phosphatases	4 to 6 hours
Alkaline phosphatases	8 to 12 hours

The vital reaction occur at the area surrounding the burns area is an important factor to determined the nature of burn, whether antemortem or postmortem.

PATHOPHYSIOLGYOF VITAL REACTION-

Dilatation of blood vessels.

⇩

Fluid leakage from blood vessels in to the tissue.

⇩

Slower blood flow

⇩

Haemoconcentration

⇩

II. Biochemical mediators released from damaged tissue.

⇩

Vasodilatation.

⇩

Release of Histamine and nitric oxide.

⇩

Arteriolar dilatation.

⇩

Opening of new capillary beds.

⇩

Increased blood flow.

⇩

Dilatation of venules for accomadation.

From the review following conclusion was arrived :

Determining the actual cause of death is a difficult one. Carboxy haemoglobin may or may not be an independent determining factor especially it's linkage to other factor concerned with antemortem burns is so strong.

From a medico legal standpoint , carboxy haemoglobin alert the forensic expert , about the aliveness of the victim at the time of fire.

Presence of carboxy haemoglobin is a definite indicator of life, at thetime of fire, therefore excludes the postmortem burns. At the same time mere absence of carbon monoxide in the blood, does not indicate postmortem burns. It should be confirmed along with other associated factors of antemortem burns.

MATERIALS AND METHODS

SETTING	:	Government Rajaji Hospital and Madurai Medical College, Madurai .
COLLABRATIVE DEPARTMENT.	:	Department of Biochemistry, Madurai Medical College, Madurai.
STUDY DESIGN	:	Prospective Qualitative Analymical Study
SAMPLE SIZE	:	100 Cases

LIMITATIONS

1. In this study all cases died of burns above the AGE of 10 years included.
2. Presence of carboxy hemoglobin only analyzed.

LABORATORY DATA

MATERIALS:- Heparinized venous blood samples taken and tested in the biochemistry department.

METHODS:- Presence of carboxy hemoglobin in the venous blood has been find out by

1. Spectroscope.
2. Chemical method.

SPECTROSCOPE:- Based on the principle, that the blood which is present in the suspected solution absorbing the rays in the spectrum, there by produce dark absorption bands of peculiar character, that vary with the type of the blood pigment.

In case of oxyhemoglobin- two distinct bands in the yellow region between **D** and **E** , nearer the **D** the band is half that of near **E**.

In case of carboxy hemoglobin the spectrum has the similar appearance that of oxyhemoglobin, but the spectrum remains unchanged, after the addition of **ammonium sulphide** which reduces oxyhemoglobin.

Figure–7:

HOPPER-SEYLER'S TEST:-(sodium hydroxide test)

One ml of blood is taken and added to the 10 ml of water and then 1 ml of 5% sodium hydroxide is added. Following coloration indicates the presence of various concentration of carboxy hemoglobin.

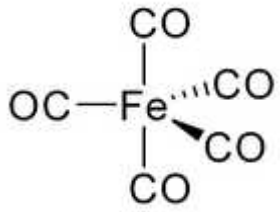
Brown color	–	Normal blood.
Straw yellow color	–	Less than 20% CoHb.
Pink color	–	Greater than 20% CoHb.

Bed side preliminary test.

Drop of blood is added to 10 to 15 ml of water and shacked well. On white back ground-pink discoloration of water-detects the presence of carbon monoxide.. Normal blood does not change in to pink.

There is no definite level that can confirm the presence or absence of carboxy hemoglobin in the blood. A level above 10% is needed to confirm the diagnosis, unless the person is a heavy smoker.

Carbon monoxide concentration in arterial blood is not significantly different from venous blood. So arterial blood does not required for diagnosis.



RESULTS

100 cases have been taken into this study and the various comparative statements are furnished below :

Table –1 : Age/ Gender Distribution

Age	Male	Female	Total
<18	2	9	**11**
18-30	10	33	**43**
31-40	14	10	**24**
41-50	4	5	**9**
>50	2	11	**13**
Total	**32**	**68**	**100**

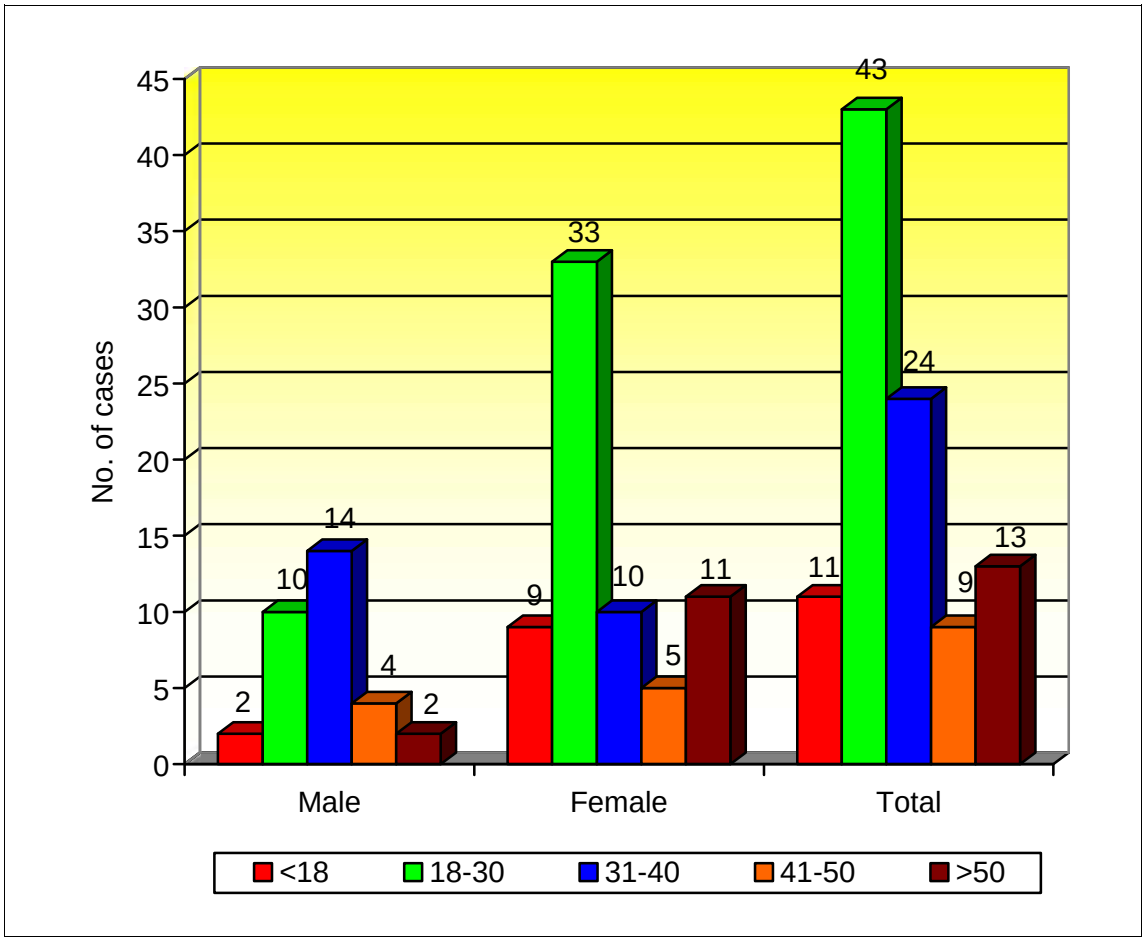
According to age concern the following details are observed:

Among the 100 burn death 11% comes under the age group of 18 years. 43% of deaths occur between 18 to 30 years; 24% occurs between 31 to 40 years. 9% occurs between 41 o 50 years and 13% of deaths occurs above 50 years of age. So the age group between 18 to 30 most commonly involved in burns. Next the 31 to 40 age group comes under

the vulnerable group. Only 13% of the old age group involved mostly because of accident.

Most commonly and highly the women under the age group of 18 to 30 are involved in burns i.e. 33%

Graph –1 : Age Gender distribution



Table–2: Gender/ Occurrence of Death

Gender	Within 24 Hrs	24 Hrs to 48 Hrs	Above 48 hrs	Total
Male	11	7	14	**32**
Female	25	5	38	**68**
Total	**36**	**12**	**52**	**100**

Among the 36 cases died within 24 hrs, 25 cases found to be female and 11 cases were male. In total in the cases which died in all the time 68% of the cases were female while 32% of the cases were males. According to our study the deaths are occurring after 48 hours.

Graph–2: Gender/ Occurrence of Death

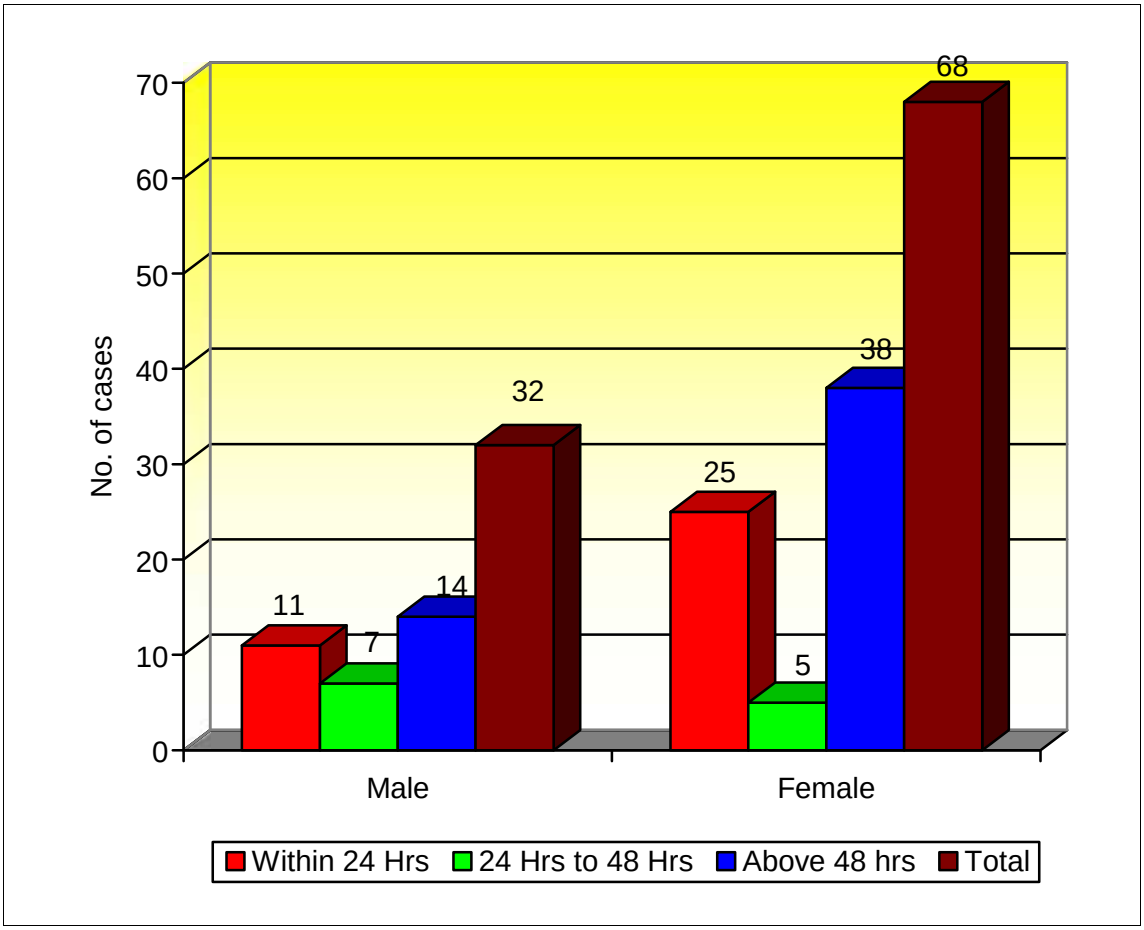


Table -3: Age Vs. Occurrence of death

Age	Within 24 Hrs	24 Hrs to 48 Hrs	Above 48 hrs	Total
<18	4	Nil	7	11
18-30	14	4	25	43
31-40	6	5	13	24
41-50	3	2	4	9
>50	9	1	3	13
Total	**27**	**11**	**51**	**89**

Regarding time of death after burn and age 100 cases died,11% of cases died was 11 to 18 years among these case 4% died within 24 hours 7% died after 48 hours.

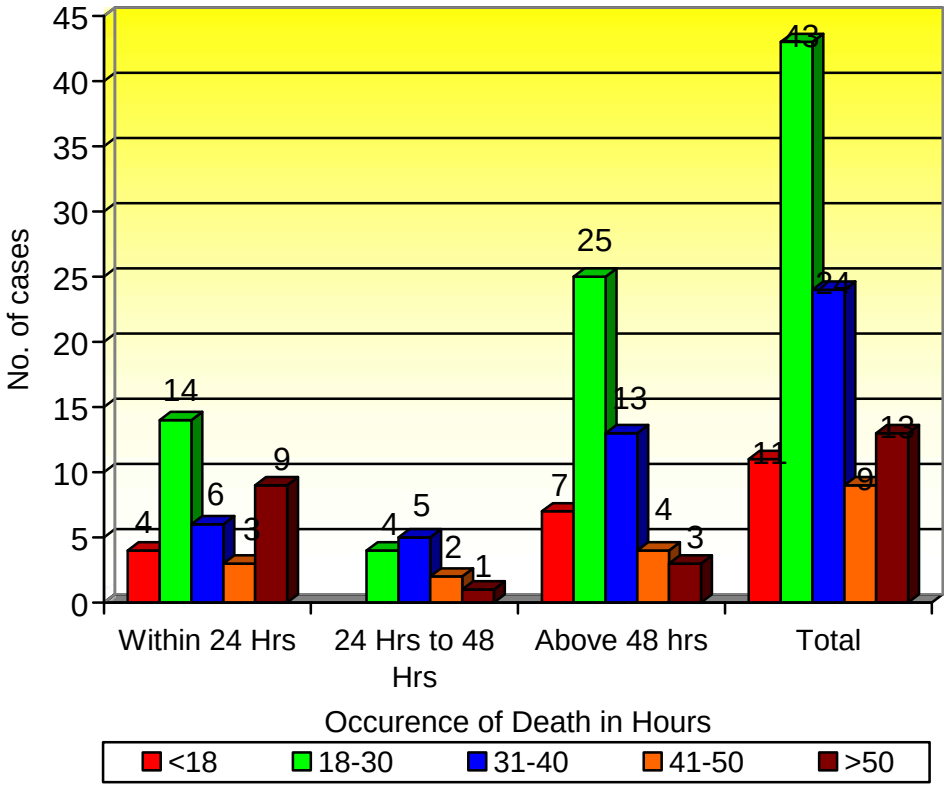
43% of cases died was between 18 to 30 years among them 14 % died within 24 hours, 4% died between 24 to 48 hours, 25% died after 48 hours.

24% of cases died was between 31 – 40 years among them 6% died within 24 hours, 5% died within 24 to 48 hours, 13% died after 48 hours.

9% of cases died was between 41 -50, among them 3% died within 24 hours, 2% died within 48 hours.

13 % of cases died was above 50 years . among them 9% died within 24 hours, 1% died between 24 to 48 hours, 3% died after 48 hours.

Graph -3: Age Vs. Occurrence of death



Table–4: Percentage of Burn Vs occurrence of Death

%age of Burn	Within 24 Hrs	24 Hrs to 48 Hrs	Above 48 hrs	**Total**
20-40	1	0	5	**6**
41-60	0	2	18	**20**
61-80	3	3	16	**22**
81-100	32	7	13	**52**
Total	36	12	52	**100**

Among BSA of 20% - 40% -- 1% of death occur within 24 hours .

No death between 24 – 48 hours.

5% of death occur after 48 hours.

Among BSA of 41% - 60% -- no death within 24 hours.

2% death between 24 to 48 hours.

18% of death after 48 hours.

Among BSA of 61% --80% -- 3% of death within 24 hours.

3% of death between 24 – 48 hours.

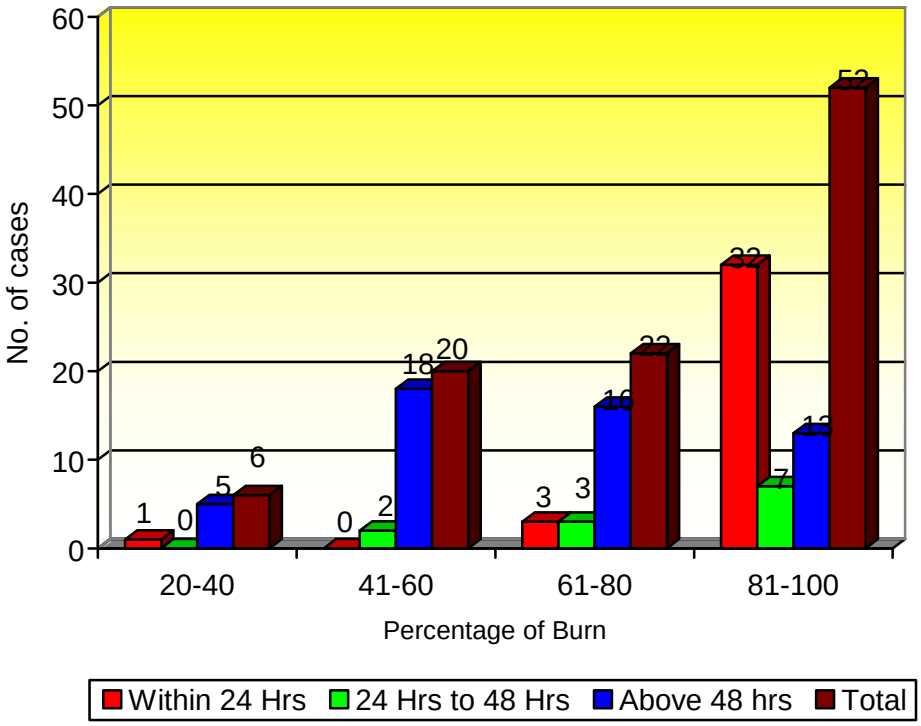
16% of death after 48 hours.

Among BSA of 81% -100% -- 32% of death within 24 hours.

7% of death between 24 – 48 hours.

13% of death after 48 hours.

Graph–4: Percentage of Burn Vs occurrence of Death



Table–5 Gender Vs Place of Occurrence

Sex	Indoor	Outdoor	Total
Male	30	2	32
Female	62	6	68
Total	92	8	100

When correlate the gender with place of occurrence.

Among 32% -- 30% of male death in indoor.

Only 2% in outdoor.

Among 68 % -- 62% of female death in indoor.

61% of female death in outdoor.

Graph–5 : Gender Vs Place of Occurrence

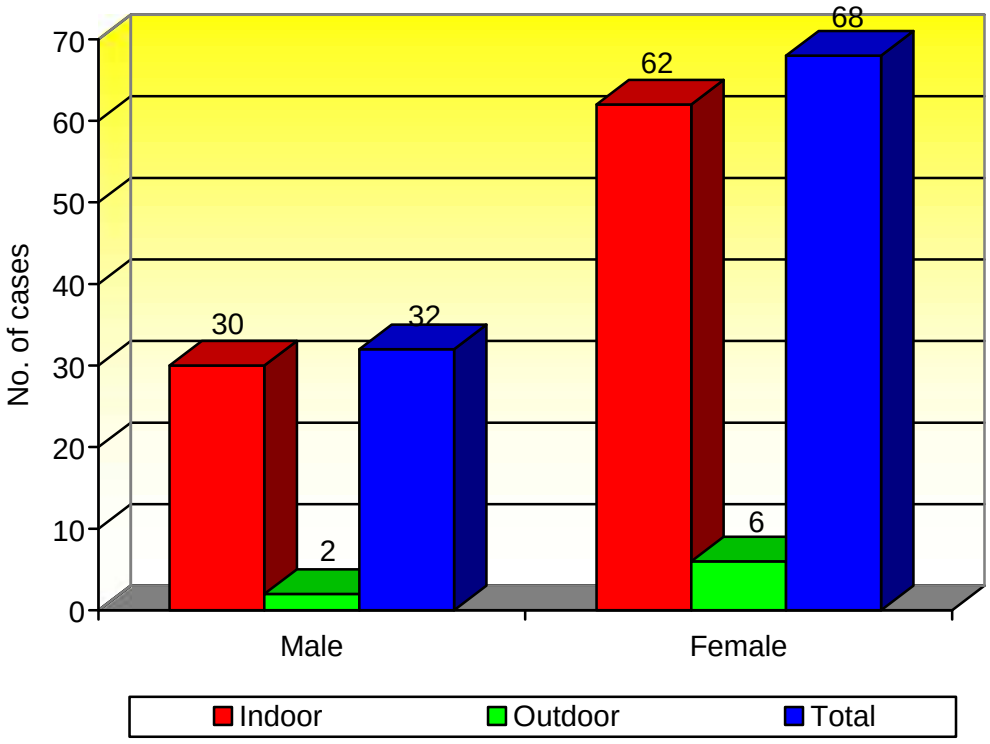
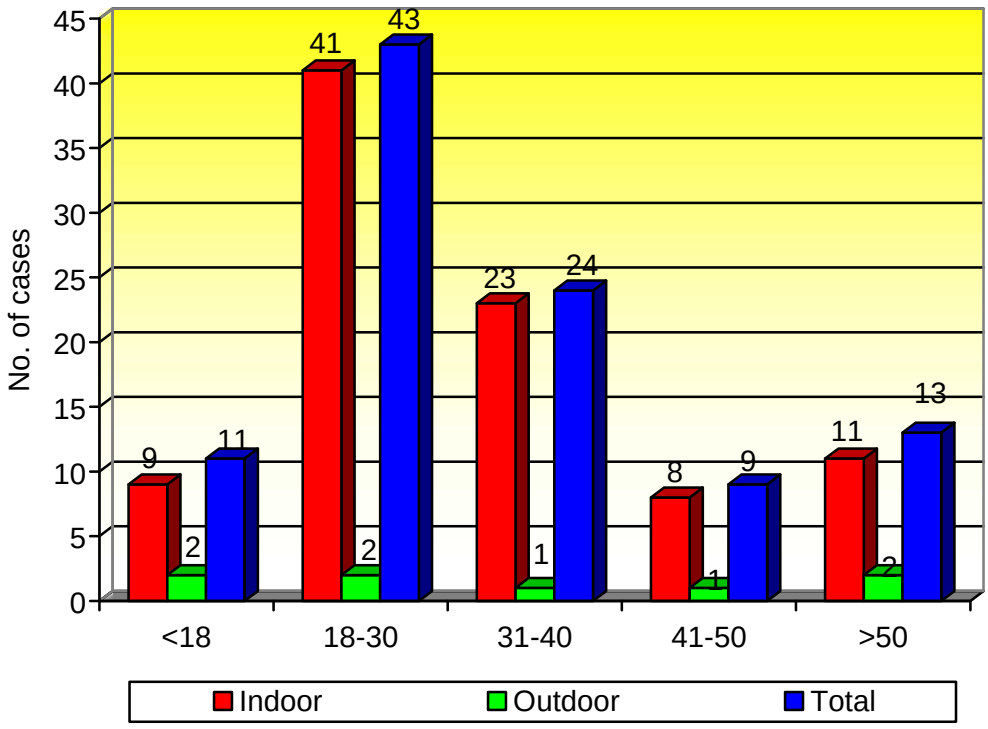


Table –6: Age Vs. Place of occurrence

Age	Indoor	Outdoor	Total
<18	9	2	11
18-30	41	2	43
31-40	23	1	24
41-50	8	1	9
>50	11	2	13
Total	92	8	100

When correlate the age with place of occurance – highest percentage 41% in indoor death was between 18 – 30 years. Next 23% within 31 – 40 years. Next 11% above 50 years.

Graph–6 : Age Vs. Place of occurrence



Table–7: Percentage of Burn Vs Place of Occurrence

%age of Burn	Indoor	Outdoor	Total
20-40	6	0	6
41-60	19	1	20
61-80	22	0	22
81-100	45	7	52
Total	92	8	100

Table–8: Percentage of Burn Vs Place of Occurrence

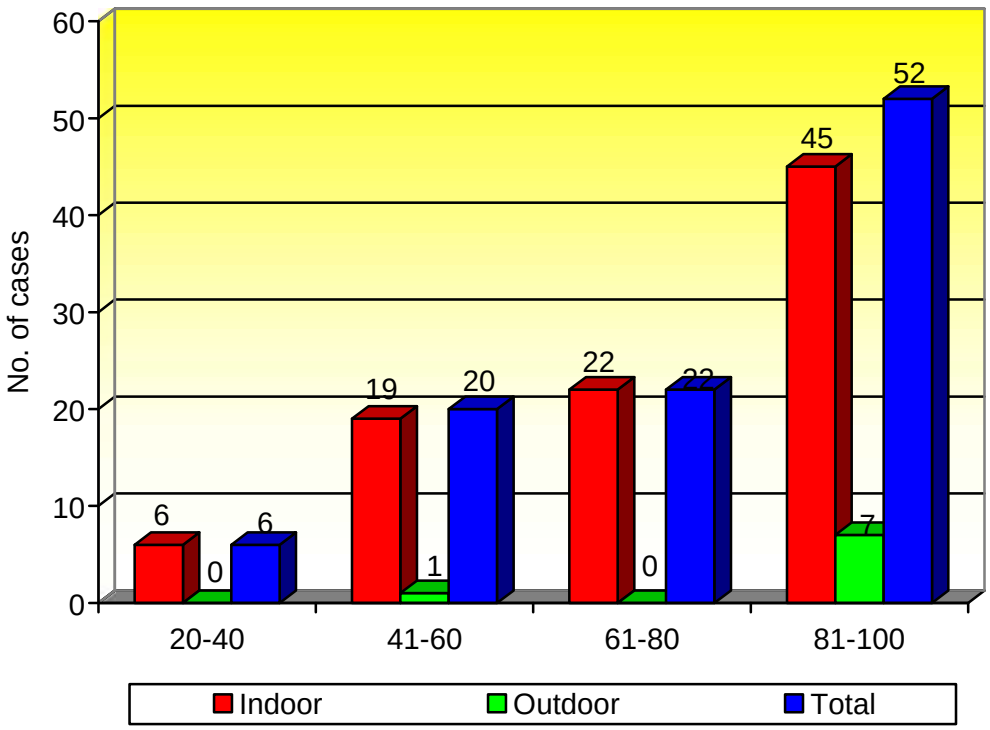
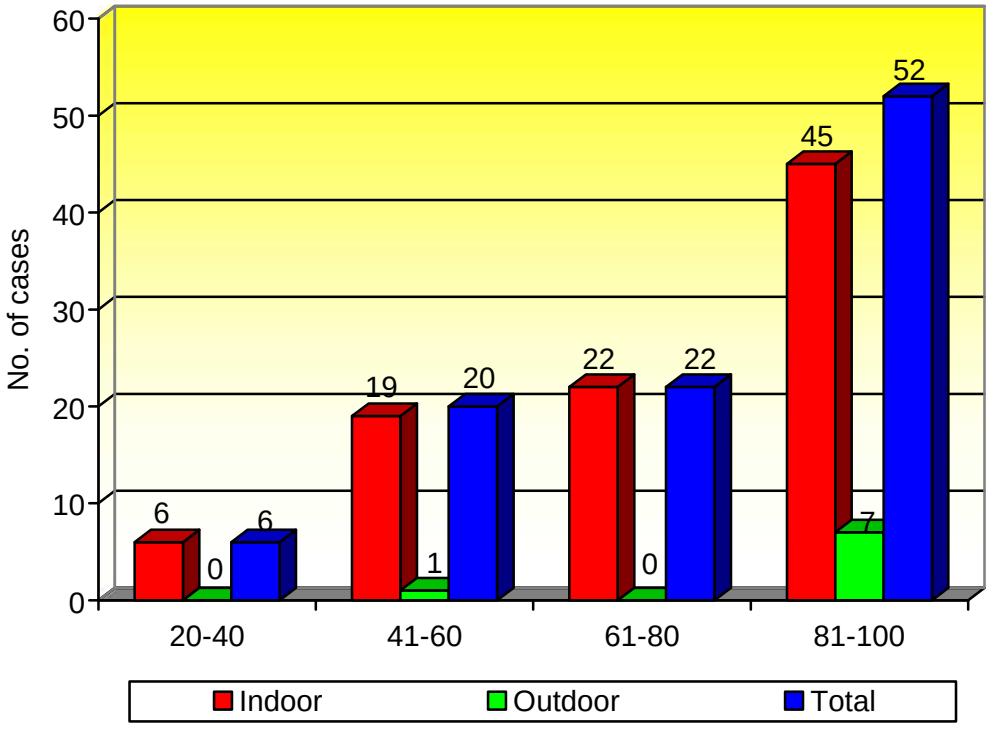


Table – 9: Percentage of Burn Vs Place of Occurrence

%age of Burn	Indoor	Outdoor	Total
20-40	6	0	6
41-60	19	1	20
61-80	22	0	22
81-100	45	7	52
Total	92	8	100

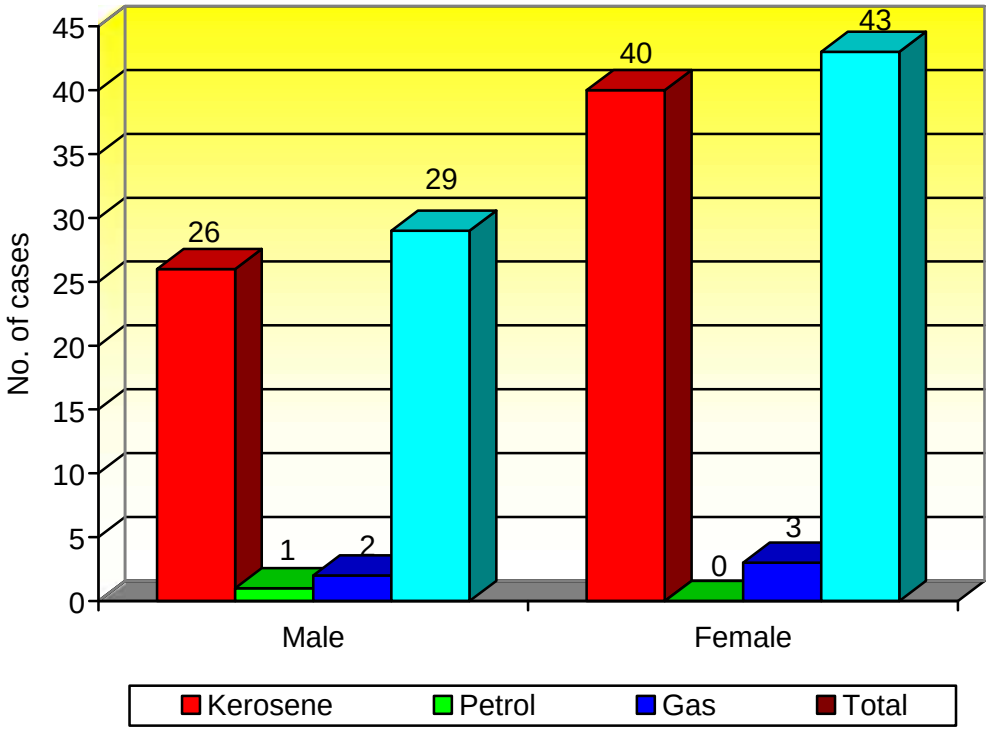
Graph–9: Percentage of Burn Vs Place of Occurrence



Table–10: Gender Vs Fire Accelerant

Gender	Kerosene	Petrol	Gas	Total
Male	26	1	2	29
Female	68	0	3	71
Total	94	1	5	100

Graph–10 : Gender Vs Fire Accelerant



Table–11 : Age Vs Fire Accelerant

Age	Kerosene	Petrol	Gas	Total
<18	7	0	1	**8**
18-30	28	0	0	**28**
31-40	19	0	2	**21**
41-50	5	0	2	**7**
>50	7	1	0	**8**
Total	**66**	**1**	**5**	**72**

Graph–11 : Age Vs Fire Accelerant

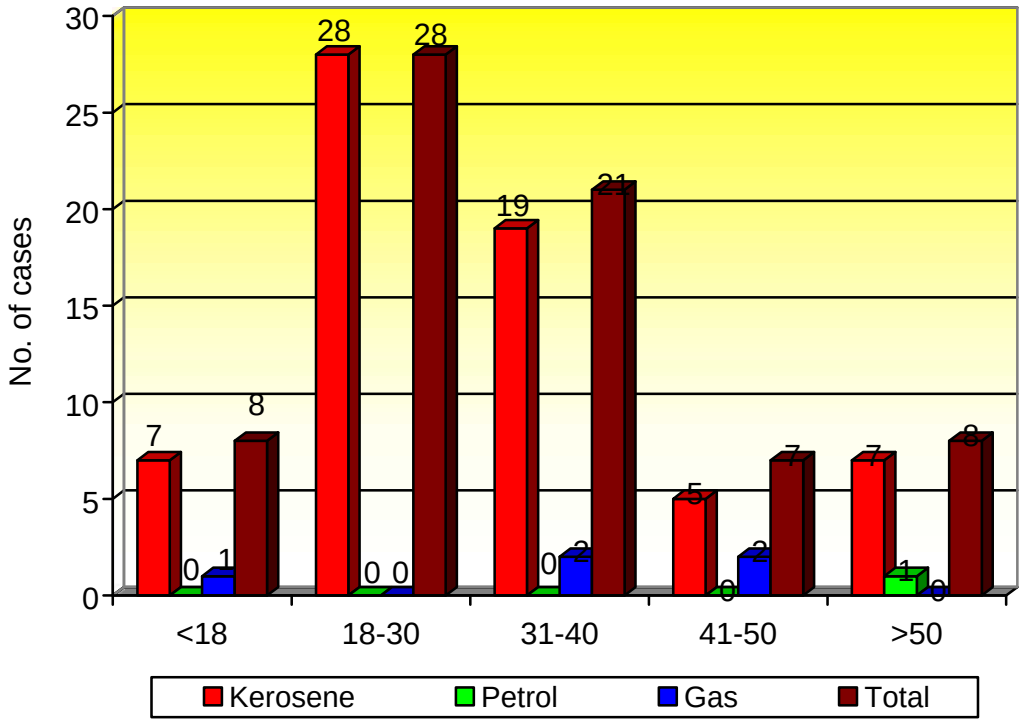
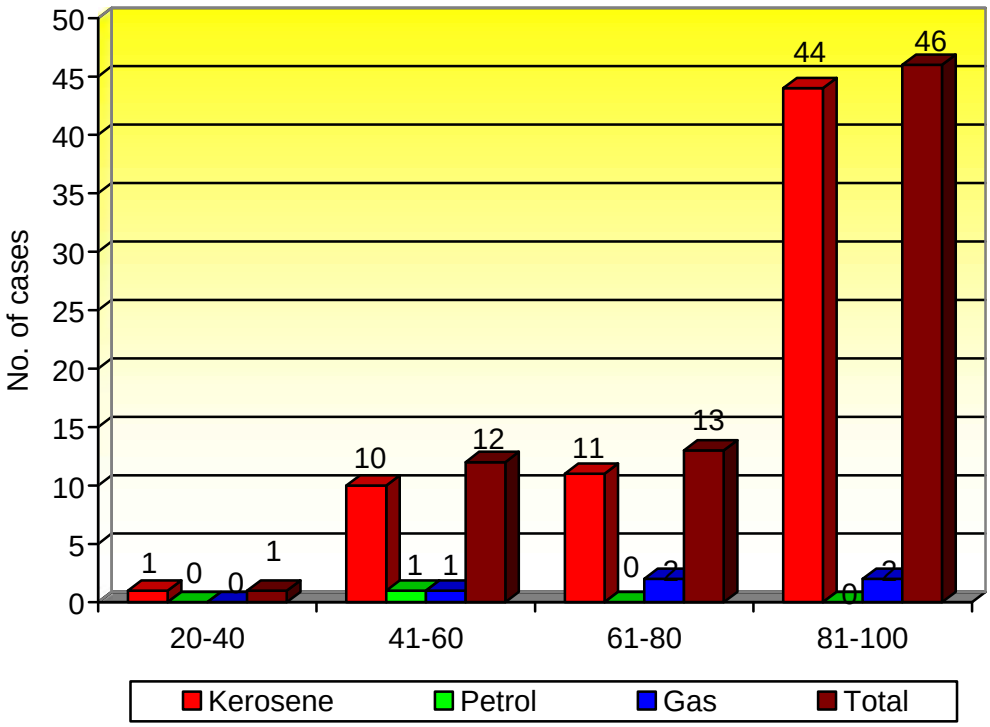


Table –12: Percentage of Burn Vs Fire Accelerant

%age of Burn	Kerosene	Petrol	Gas	Total
20-40	1	0	0	**1**
41-60	10	1	1	**12**
61-80	11	0	2	**13**
81-100	44	0	2	**46**
Total	**66**	**1**	**5**	**72**

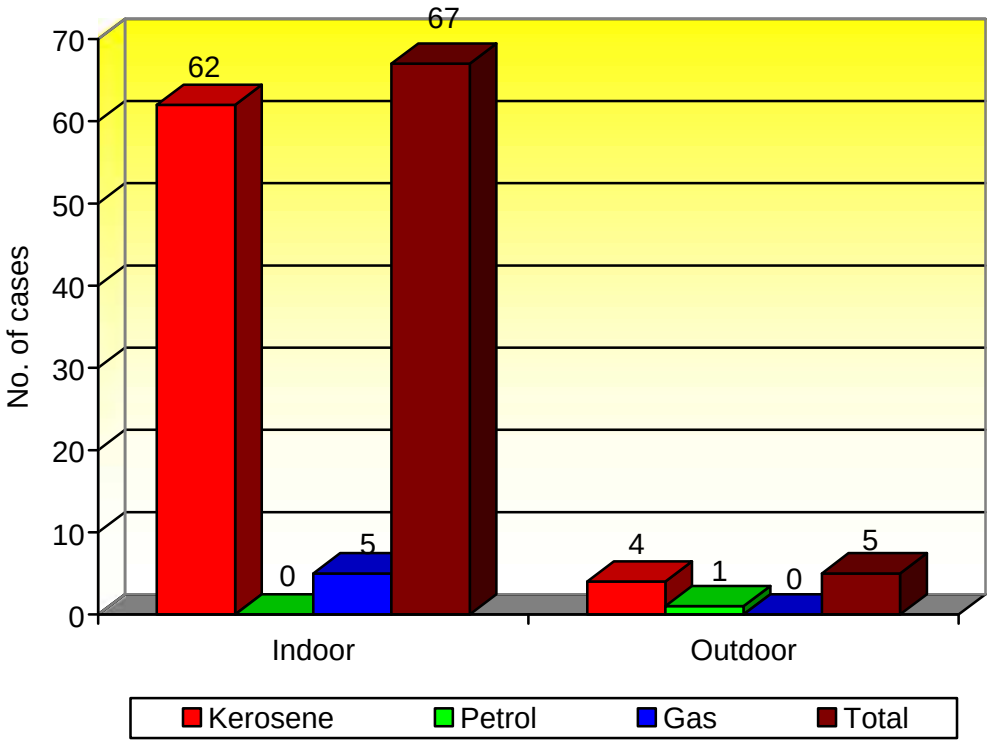
Graph –12: Percentage of Burn Vs Fire Accelerant



Table–13 : Place of occurrence Vs Fire Accelerant

Place of occurrence	Kerosene	Petrol	Gas	Total
Indoor	62	0	5	67
Outdoor	4	1	0	5
Total	**66**	**1**	**5**	72

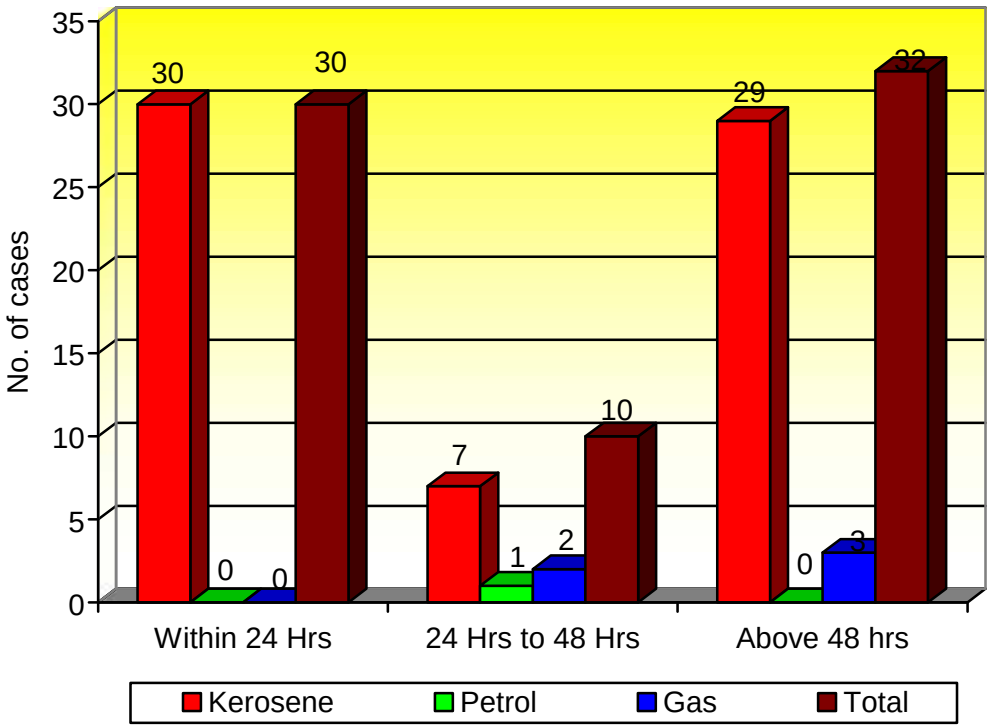
Graph–13 : Place of occurrence Vs Fire Accelerant



Table–14 : Fire accelerant Vs Death interval

Death interval	Kerosene	Petrol	Gas	Total
Within 24 Hrs	30	0	0	**30**
24 Hrs to 48 Hrs	7	1	2	**10**
Above 48 hrs	29	0	3	**32**
Total	**66**	**1**	**5**	**72**

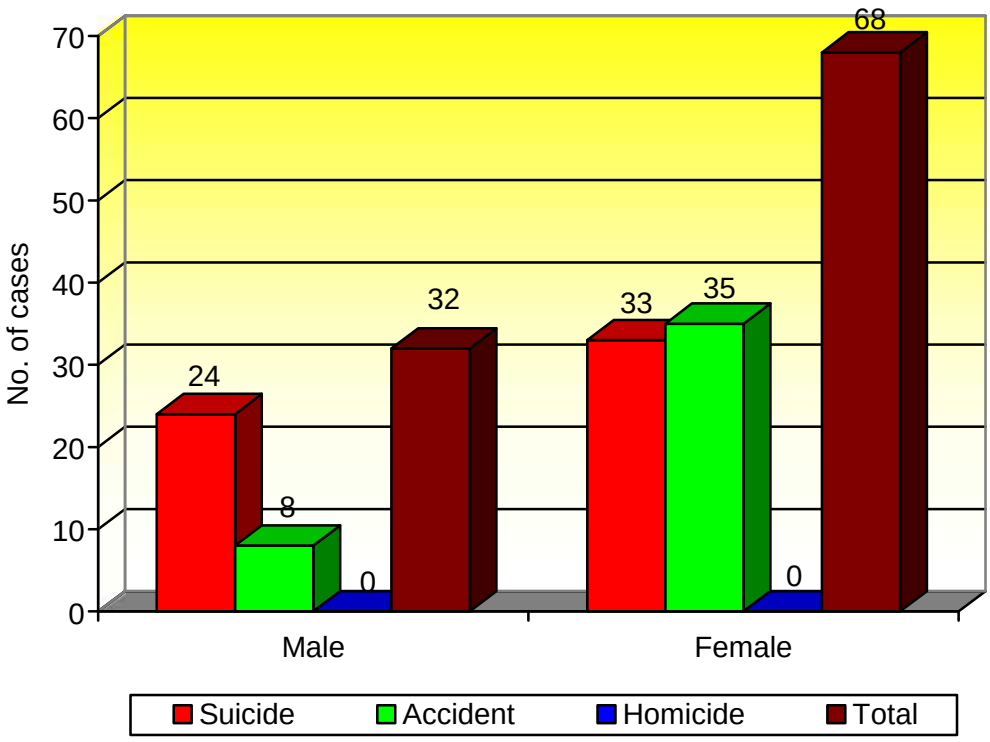
Graph–14 : Fire accelerant Vs Death interval



Table–15 : Gender Vs Manner of death

Gender	Suicide	Accident	Homicide	Total
Male	24	8	0	32
Female	33	35	0	68
Total	**57**	**43**	**0**	**100**

Graph–15 : Gender Vs Manner of death



Table–16 : Age Vs Manner of death

Age	Suicide	Accident	Homicide	Total
<18	5	6	0	**11**
18-30	25	18	0	**43**
31-40	18	6	0	**24**
41-50	5	4	0	**9**
>50	4	9	0	**13**
Total	**57**	**43**	**0**	**100**

Graph–16 : Age Vs Manner of death

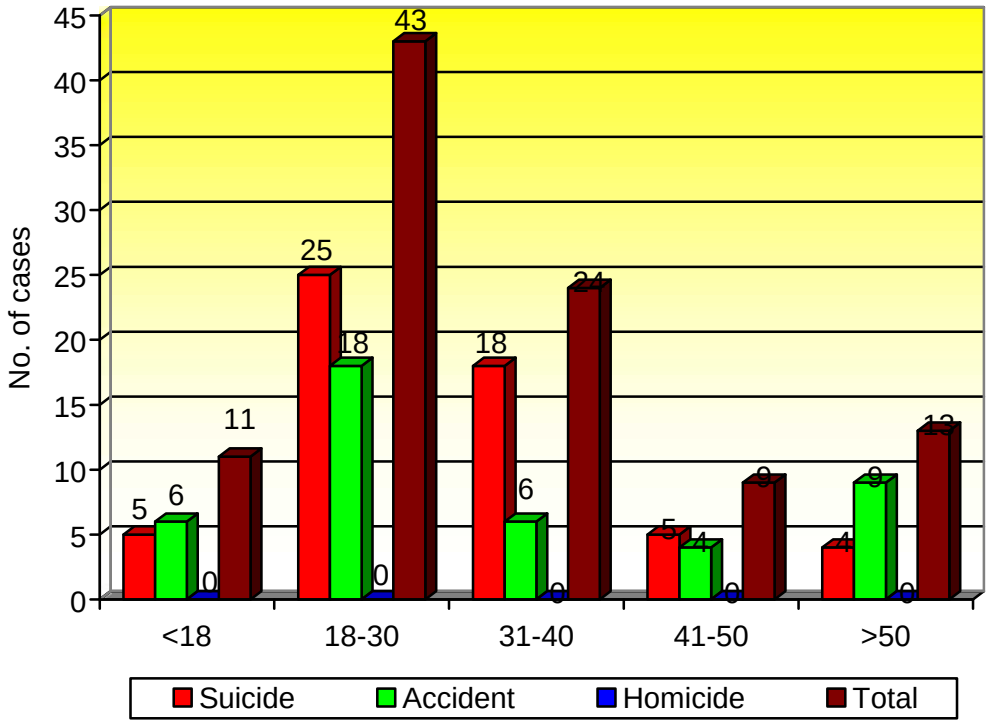
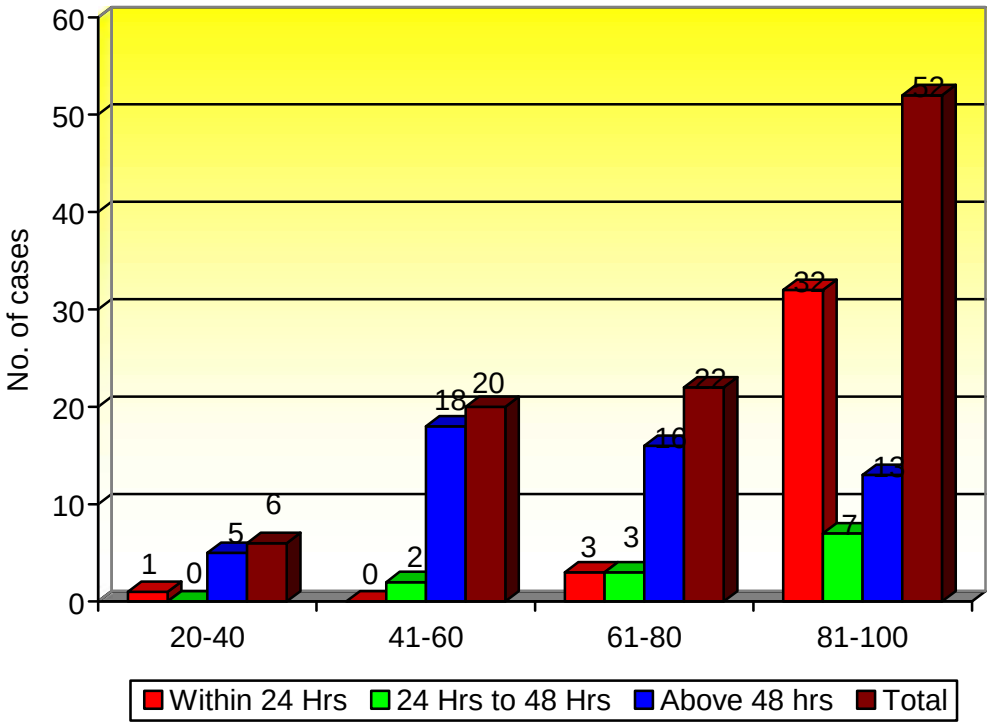


Table –17: Percentage Vs Manner of death

%age of Burn	Suicide	Accident	Homicide	Total
20-40	0	6	0	**6**
41-60	9	11	0	**20**
61-80	9	13	0	**22**
81-100	39	13	0	**52**
Total	**57**	**43**	**0**	**100**

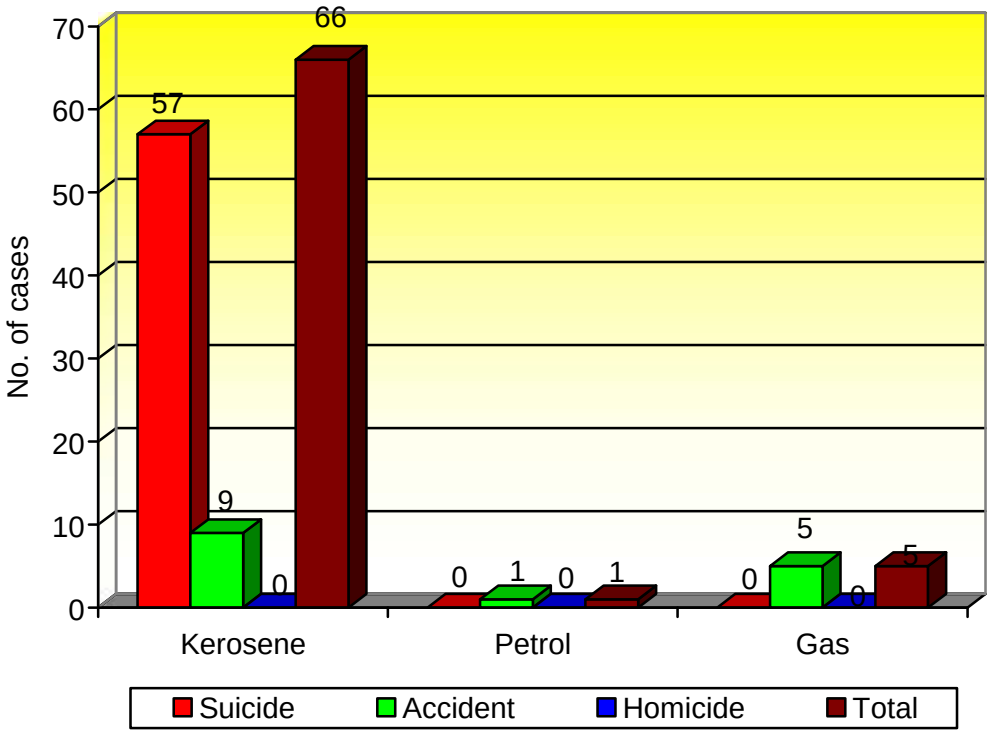
Graph –17: Percentage Vs Manner of death



Table–18: Manner of Death Vs Fire Accelerant

	Suicide	Accident	Homicide	Total
Kerosene	57	37	0	**66**
Petrol	0	1	0	**1**
Gas	0	5	0	**5**
Total	**57**	**43**	**0**	**72**

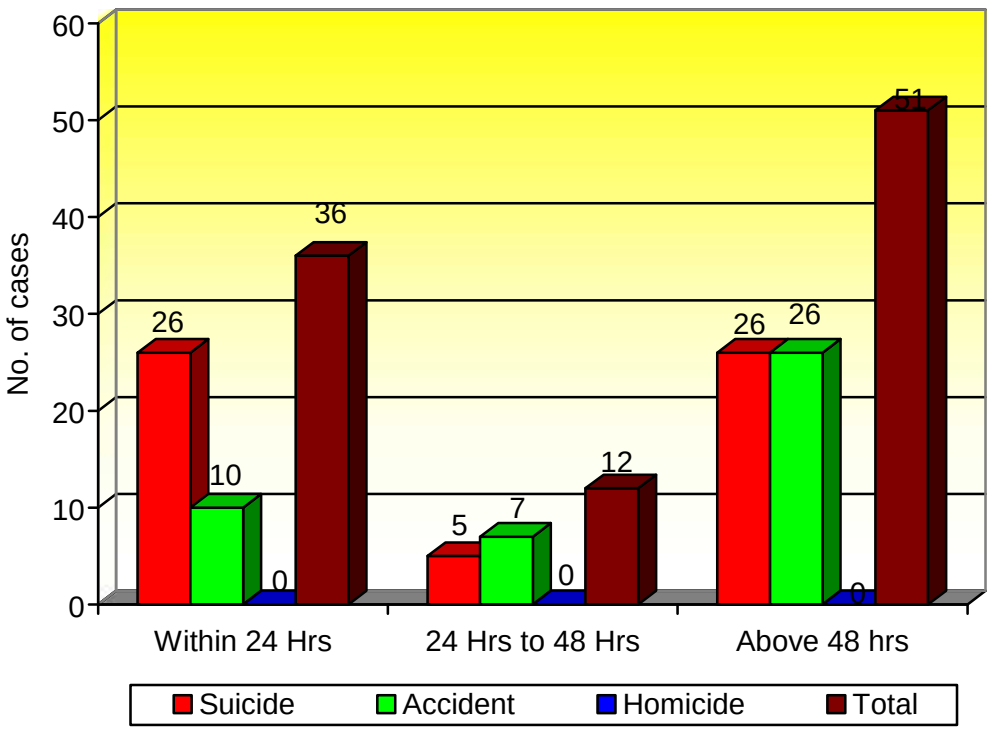
Graph–18: Manner of Death Vs Fire Accelerant



Table–19: Manner of Death Vs Occurrence of Death

Occurrence of death	Suicide	Accident	Homicide	Total
Within 24 Hrs	26	10	0	36
24 Hrs to 48 Hrs	5	7	0	12
Above 48 hrs	26	26	0	51
Total	**57**	**43**	**0**	**100**

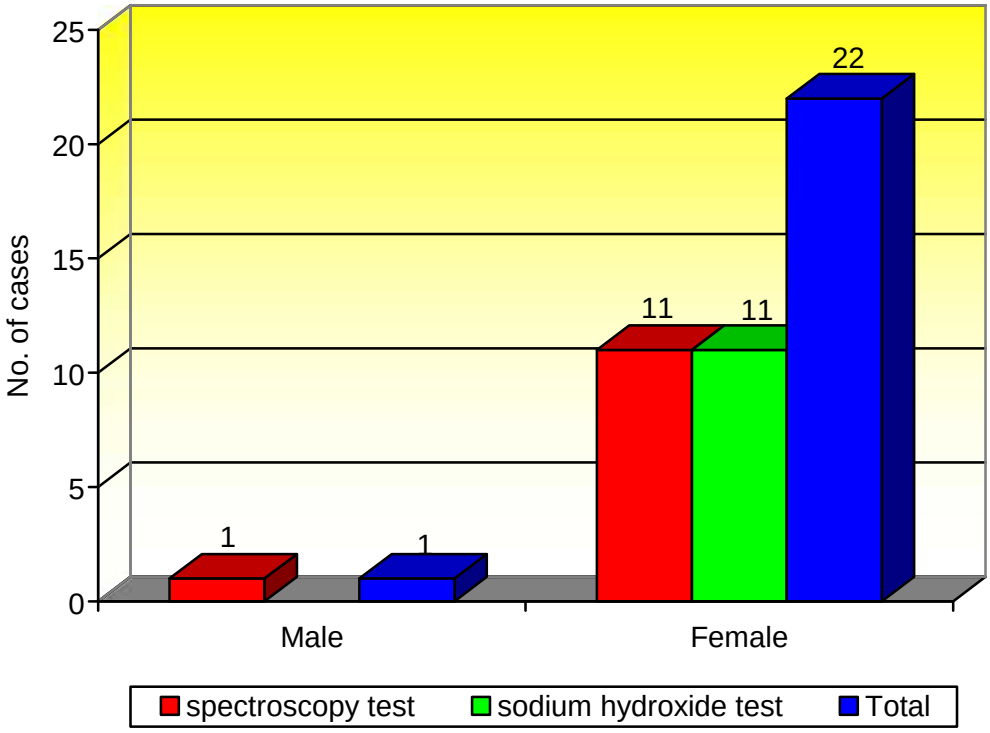
Graph–19: Manner of Death Vs Occurrence of Death



Table–20 : Gender Vs Test

Gender	Spectroscopy test	Sodium hydroxide test	Total
Male	1	1	1
Female	11	11	11
Total	**12**	**12**	**12**

Table–20 : Gender Vs Test



Table–21 : Age Vs test

Age	Spectroscopy Test	Sodium hydroxide Test	Total
<18	1	1	**1**
18-30	5	5	**5**
31-40	2	2	**2**
41-50	2	2	**2**
>50	2	2	**2**
Total	**12**	**12**	**12**

\

Graph–21 : Age Vs test

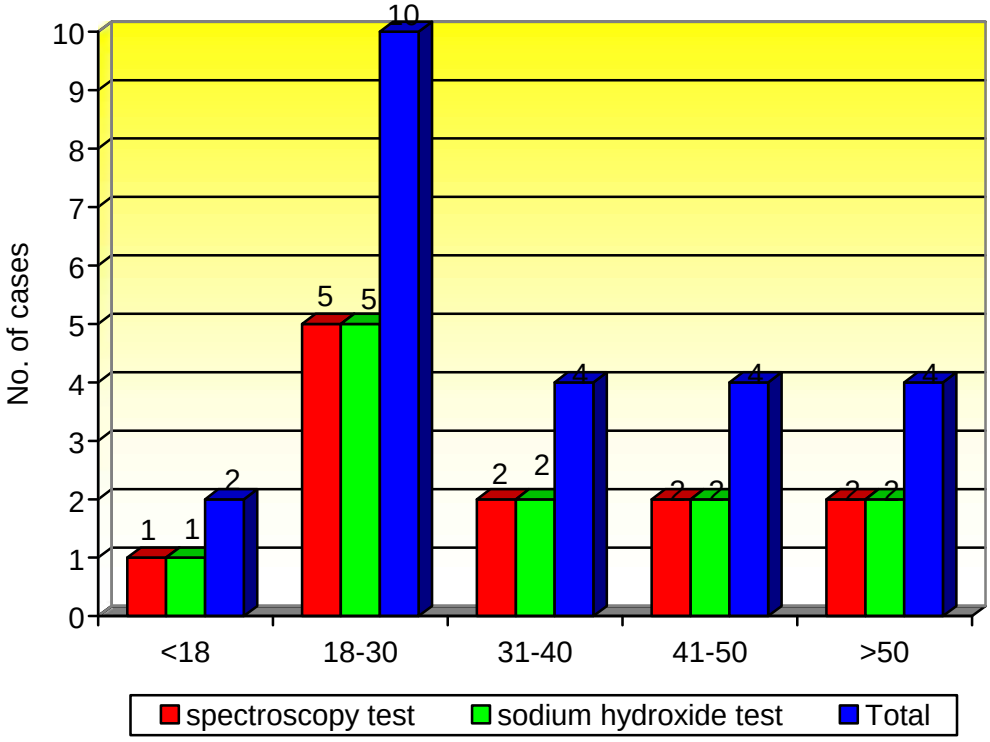


Table –22: Percentage of Burn Vs Fire Accelerant

%age of Burn	Spectroscopy test	Sodium hydroxide test	Total
20-40	3	3	**3**
41-60	2	2	**2**
61-80	3	3	**3**
81-100	4	4	**4**
Total	**12**	**12**	**12**

Graph –22: Percentage of Burn Vs Fire Accelerant

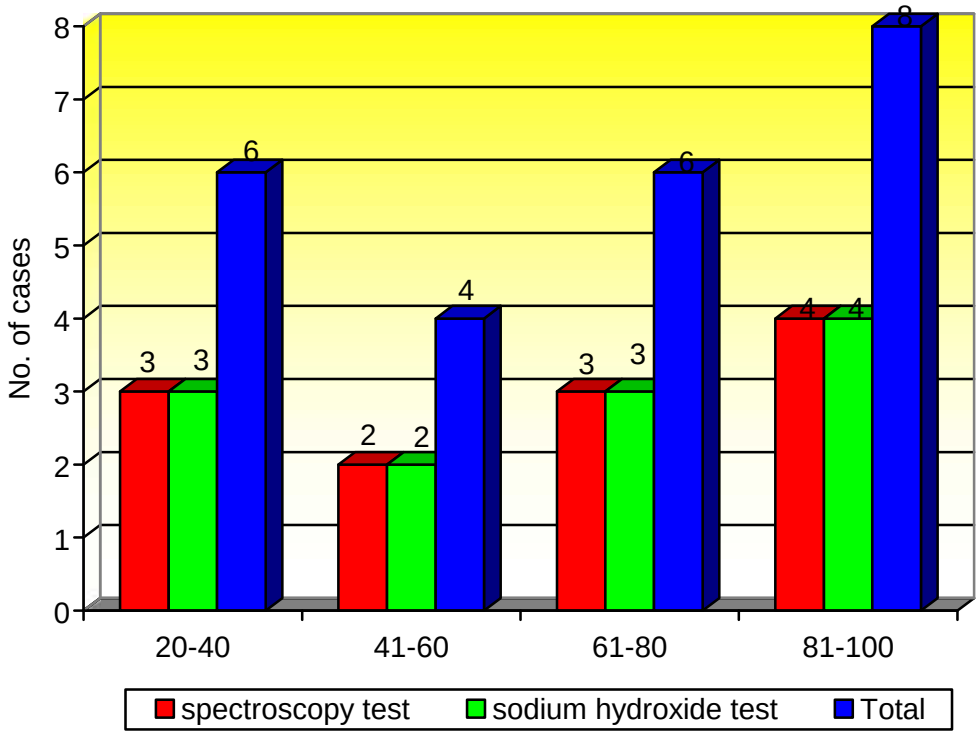


Table – 23 : Place of occurrence Vs Test Result

Place of occurrence	Spectroscopy test	Sodium hydroxide test	Positive result for CoHb
Indoor	12	12	12
Outdoor	0	0	0
Total	**12**	**12**	**12**

Test results are positive only in indoor death.

Table–24 : Test Result Vs Occurrence of Death

Occurrence of death	Spectroscopy test	Sodium hydroxide test	Total
Within 24 Hrs	4	4	8
24 Hrs to 48 Hrs	1	1	2
Above 48 hrs	7	7	14
Total	**12**	**12**	**24**

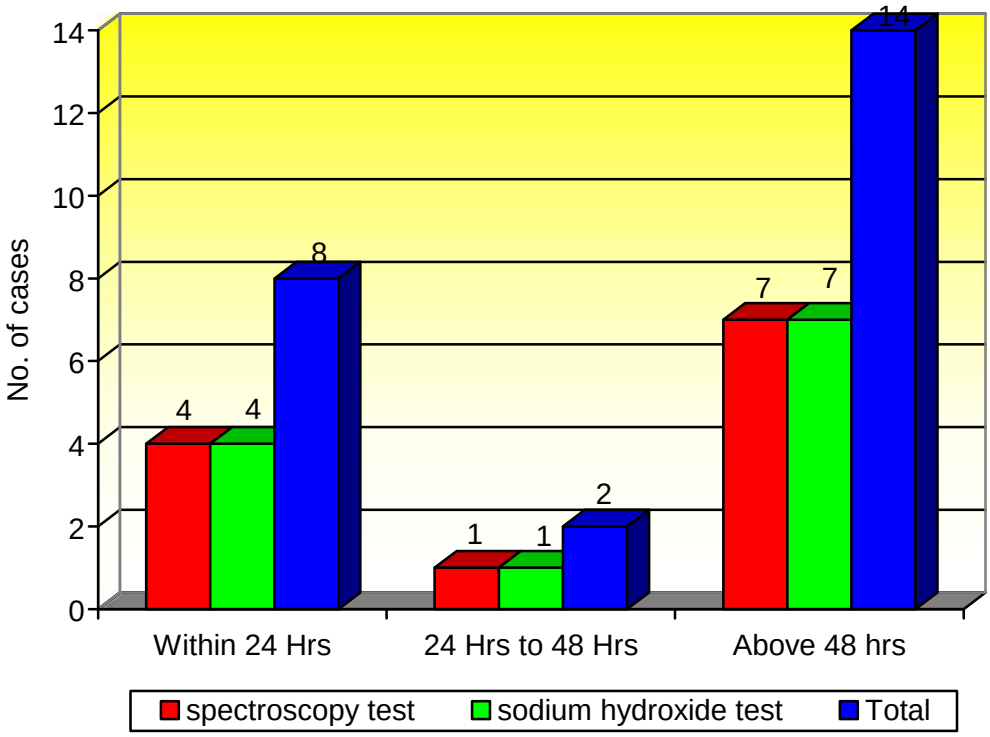
Among the 100 cases

4% of test results are positive – within 24 hours.

1% between 24 – 48 hours.

7% above 48 hours.

Graph–23 : Test Result Vs Occurrence of Death



Table–25 : Test Result Vs Fire Accelerant

%age of Burn	Spectroscopy test	Sodium hydroxide test	Positive result for CoHb
Kerosene	12	12	**12**
Petrol	0	0	**0**
Gas	0	0	**0**
Total	**12**	**12**	**12**

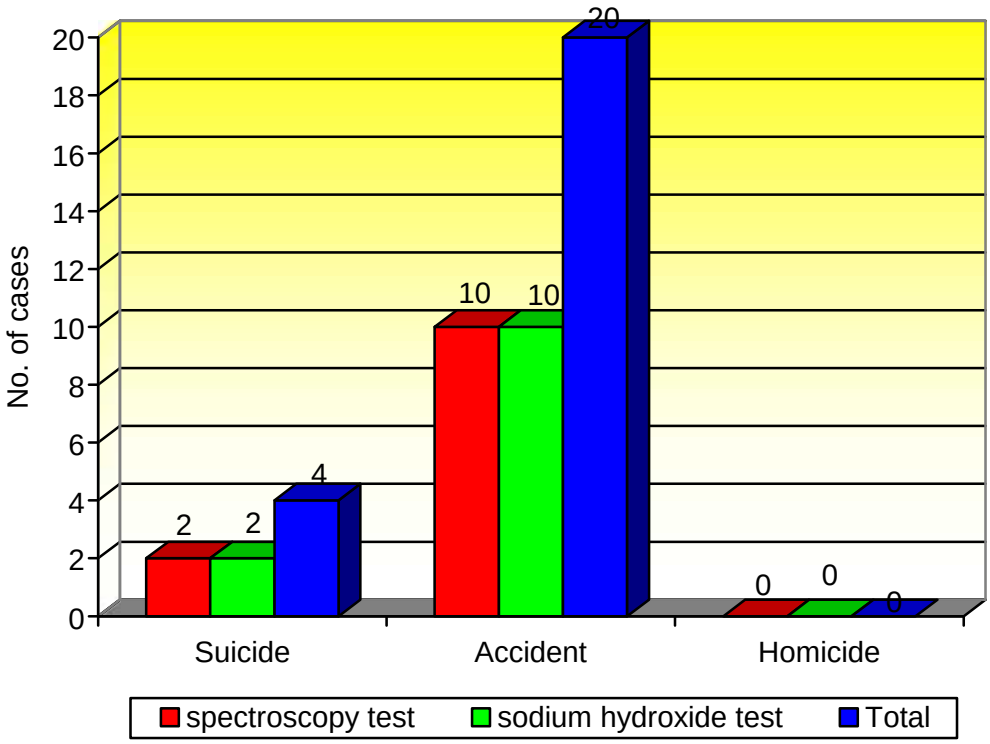
Test results are positive all the cases in which kerosene used as the fire accelerant.

Table–26 :Test Result Vs Manner of Death

%age of Burn	**Spectroscopy test**	**Sodium hydroxide test**	Total
Suicide	2	2	**2**
Accident	10	10	**10**
Homicide	0	0	**0**
Total	**12**	**12**	**12**

Test resuls are positive for CoHb in 2% of the suicide case , among 57%, 10% of the , accident case among 43%.

Graph–24 :Test Result Vs Manner of Death



DISCUSSION

Death due to burn is one of the most important common cause for suicide especially in women. In some of the dowry death case, the women may be murdered and burned.

The analysis of carboxy haemoglobin in the blood is essential for diagnosis of carbon monoxide poisoning. It is useful to differentiate the antemortem burns from postmortem burns .It indicates the inhalation of smoke before death and so the person was alive at the time of burns. The most accurate method for measuring carbon monoxide in blood, was gas chromatography and spectrophotometry.

In this study relation between blood carboxy haemoglobin and age, sex, fire accelerant and circumstantial evidence analyzed by spectroscopy and sodium hydroxide test which are available in our hospital.

Previous analysis of data,linked the presence of carboxy haemoglobin with antemortem burn only. In postmortem burns it is absent in all cases.

Previous studies from Yoshida et al (1991) published the data from, 120 house fire victim in Japan,in which, only in nine people the presence of carboxy haemoglobin noted.

Nature of variability in carbon monoxide may be illustrated by the fact, the two bodies burned in a same house, lying side by side may have widely varying CoHB level in blood.

In our study, of the 100 cases of burn death, the carboxy haemoglobin is present only in 12 cases, which account for 12%.

In our study all the 12 cases that show carboxy haemoglobin in the blood were died in a closed space.The observed differences in the presence of CoHb in our study regarding to the place of occurrence,were statistically significant and also it strongly supports the scientific fact that Co poisoning occurs most commonly in closed space.

Fire accelerant used was kerosene .Soot particles and vital reaction present in all this cases. Exposure time to fire is prolonged in all cases.

In this study in all the 12 cases presence of carboxy haemoglobin correlate with pinkish discoloration of internal organ and presence of soot particle.

On the otherhand,the relative difference in CoHb in antemortem burns indicates, CoHb alone does not qualify as a predictive marker for antemortem burns.

Neverthless ,we hope that the presented data add to a more complete understanding of the characteristics of CoHb in antemortem burns.

SUMMARY

With certain criteria 100 cases are selected carefully and evaluated on forensic and laboratory aspect after institutional ethical clearance with on informed consent. The presence of carboxy hemoglobin in hundred cases of, death due to burns analyzed with spectroscopy test and Hopper seyler's test .

The venous blood that is collected directly from right side of the heart and then heparin added. The sample handed over to the biochemistry department for analysis.

By enquiry and with the inquest report, the particulars are collected and correlated with the presence of carboxy hemoglobin.

The particulars collected are age, sex, percentage of burns, death interval, circumstantial evidence, fire accelerants used, and manner of death. The data were entered in computer and analyzed statistically.

There were 32 males and 68 females.

Out of these 100 cases , 12% of the case shows presence of carboxy haemoglobin .

Among the 12% of cases, manner of death is, accident in 10% cases and suicide in only 2% of cases.

Among the 12% of case ,in all the cases, the death occurred in closed space.

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The presence of carboxy haemoglobin significantly found more in the accidental cases and also in suicidal case, died in a closed room (indoor).

The factors that contributing for the appearance of carboxy haemoglobin in the blood are , source of carbon monoxide from the burning material ,concentration of carbon monoxide in the inhaled air , respiratory status and activity of the victim at the time of firing. Local variation in draughts and levels above the floor may also account for.

Presence of carboxy haemoglobin is a definite indicator of life at the time of fire, therefore exclude the postmortem burns .At the same

time mere absence of carbon monoxide in the blood , does not indicate post mortem burns. It should be confirmed along with the, other associated factors of antemortem burns.

CONCLUSION

- Presence of carboxy hemoglobin detected only in 12 % of the person died due to burn.

- Significantly, present only in indoor burns.

- Presence of Blood carboxy hemoglobin associated with pinkish discoloration of internal organs

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- Age, sex , percentage of burns does not influence the carboxy haemoglobin.

- Death interval and manner of death also not correlate with COHB.
- Presence of soot particles does always associated with presence of carboxy hemoglobin.

ANNEXURE

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MASTER CHART

Serial No	Sex	Age	Percentage of burns	Death Occurs			Place of Occurance		Fire Accelerant used			Manner of Death			Spectroscopy test	Sodium hydroxide test
				With in 24 hours	24 to 48 hrs	More than 48 hrs	Indoor	Out door	kerosene	Petrole	Gas	Suicide	Accident	Homicide		
1	M	35	100%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
2	F	26	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	Yes	Yes
3	M	38	90%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
4	M	32	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	Yes	Yes
5	F	75	90%	Yes	No	No	Yes	No	Yes	No	No	No	Yes	No	No	No
6	F	75	90%	Yes	No	No	Yes	No	No	No	No	No	Yes	No	No	No
7	F	60	100%	Yes	No	No	No	Yes	No	No	No	No	Yes	No	No	No
8	F	18	55%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
9	F	25	50%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
10	F	40	80%	No	Yes	No	Yes	No	No	No	No	No	Yes	No	Yes	Yes
11	F	18	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
12	F	20	70%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
13	M	35	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
14	F	25	50%	No	Yes	No	Yes	No	Yes	No	No	Yes	No	No	No	No
15	F	35	100%	No	Yes	No	Yes	No	No	No	Yes	No	Yes	No	No	No
16	M	45	100%	No	Yes	No	Yes	No	No	No	Yes	No	Yes	No	No	No
17	F	24	70%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	Yes	Yes
18	F	32	70%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
19	M	45	65%	No	No	Yes	Yes	No	No	No	Yes	No	Yes	No	No	No
20	M	40	60%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
21	F	72	40%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
22	F	16	50%	No	No	Yes	No	Yes	No	No	No	No	Yes	No	No	No
23	F	16	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No

24	M	33	70%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
25	F	19	90%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
26	M	38	90%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
27	F	38	72%	No	No	Yes	Yes	No	No	No	Yes	No	Yes	No	No	No
28	F	29	50%	No	No	Yes	Yes	No	Yes	No	No	No	Yes	No	No	No
29	F	12	65%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
30	M	68	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
31	M	63	60%	No	Yes	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No
32	F	30	80%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
33	F	50	30%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	Yes	Yes
34	F	17	40%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
35	F	21	80%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
36	M	28	50%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
37	M	38	90%	No	Yes	No	Yes	No	Yes	No	No	Yes	No	No	No	No
38	F	13	90%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
39	F	45	60%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	Yes	Yes
40	F	70	50%	No	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
41	F	40	70%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
42	M	50	90%	No	Yes	No	No	Yes	Yes	No	No	Yes	No	No	No	No
43	M	19	65%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
44	F	45	90%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
45	M	48	90%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
46	M	17	70%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
47	F	80	100%	Yes	No	No	Yes	No	Yes	No	No	No	Yes	No	Yes	Yes
48	F	15	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
49	F	22	60%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
50	F	20	50%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
51	F	20	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
52	F	23	60%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No

53	F	24	80%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
54	F	25	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
55	F	26	90%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
56	F	33	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
57	F	26	60%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	Yes	Yes
58	M	25	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
59	F	22	80%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
60	F	27	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
61	F	42	95%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
62	M	32	50%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
63	M	28	90%	Yes	No	No	Yes	No	No	No	No	No	Yes	No	No	No
64	F	85	100%	Yes	No	No	No	No	No	No	No	No	Yes	No	Yes	Yes
65	F	27	85%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
66	F	29	20%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	Yes	Yes
67	F	35	80%	No	Yes	No	Yes	No	No	No	No	No	Yes	No	No	No
68	F	18	85%	No	No	Yes	No	No	Yes	No	No	Yes	No	No	No	No
69	M	28	90%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
70	F	80	36%	Yes	No	No	Yes	No	No	No	No	No	Yes	No	No	No
71	F	20	100%	No	No	Yes	No	No	Yes	No	No	Yes	No	No	No	No
72	F	81	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
73	F	31	95%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
74	M	37	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
75	M	35	90%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
76	F	35	80%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
77	M	30	85%	Yes	No	No	Yes	No	No	No	No	No	Yes	No	No	No
78	F	18	60%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
79	M	24	90%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
80	F	30	75%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	Yes	Yes
81	F	19	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No

82	M	30	55%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
83	F	16	90%	No	No	Yes	No	Yes	Yes	No	No	Yes	No	No	No	No
84	M	33	50%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
85	M	40	90%	No	Yes	No	Yes	No	Yes	No	No	Yes	No	No	No	No
86	F	15	50%	No	No	Yes	Yes	No	No	No	Yes	No	Yes	No	No	No
87	F	28	60%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
88	F	30	70%	No	No	Yes	Yes	No	No	No	No	No	Yes	No	No	No
89	F	44	90%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
90	F	57	80%	No	No	Yes	Yes	No	Yes	No	No	Yes	No	No	No	No
91	F	17	40%	No	No	Yes	Yes	No	Yes	No	No	No	Yes	No	Yes	Yes
92	M	17	100%	Yes	No	No	Yes	No	Yes	No	No	No	Yes	No	No	No
93	M	18	100%	No	Yes	No	Yes	No	Yes	No	No	No	Yes	No	No	No
94	M	21	90%	No	Yes	No	Yes	No	Yes	No	No	Yes	No	No	No	No
95	F	30	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
96	F	33	74%	No	No	Yes	Yes	No	Yes	No	No	No	Yes	No	No	No
97	F	53	90%	Yes	No	No	Yes	No	Yes	No	No	No	Yes	No	No	No
98	F	29	80%	No	Yes	No	Yes	No	Yes	No	No	No	Yes	No	No	No
99	F	26	100%	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	No
100	M	33	95%	No	No	Yes	No	Yes	Yes	No	No	Yes	No	No	No	No